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The Roentgen Diagnosis of Pulmonary Infarcts[1]

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THE ROENTGEN diagnosis of a pulmonary infarct is probably subject to a greater percentage of error than that of any other lesion of the lung. The error is almost always a "negative" one, that is, failure to suspect an infarct as the cause of the abnormal shadow present on the roentgenogram. In its protean manifestations this pathologic process may mimic almost any other lung disease. This factor and the supposed rarity of the disease probably account for the high percentage of "negative" errors.

This paper is based, in part, on 344 instances of aseptic hemorrhagic infarction of the lungs seen at autopsy in a ten-year period (1930–39) at the Cleveland City Hospital. In 174, the infarct was the major cause of death or was an important contributory factor, but in only 22 per cent was the correct diagnosis made. In 73 cases adequate clinical and/or roentgenologic correlation with the autopsy findings was possible. A study of this series, stressing the clinical aspects, has been reported (7). In the course of that study, a number of patients were seen who recovered from a pulmonary infarct, and since that time several others, including some with various complications, have also been followed. These two groups have also been studied in the preparation of this report.

A brief review of the clinical findings will be presented, the roentgen signs will be discussed in detail and illustrated, and the differential diagnosis considered. It is hoped that, in this manner, a clearer concept of the entire picture will be gained and that the number of diagnostic errors will be reduced. If the roentgenologist is able to make a correct diagnosis of an infarct of the lung, he will have rendered the clinician a real service because of the grave prognostic import of such a diagnosis.

In recent years the differentiation of infarcts from the pneumonias has become more important, since treatment with the sulfa drugs has lowered the mortality in the latter group.

REVIEW OF THE LITERATURE

The first definite papers on the subject of roentgen diagnosis of pulmonary infarction appeared about twenty years ago. In 1922 Wharton and Pierson (12) reported 9 cases, all of which followed abdominal surgery. They described clouding of the costophrenic angle as the earliest sign and stated that the shadows of the infarct, when seen, were sharply defined.

In 1923, Wessler and Jaches (10) stated that large triangular shadows with the base toward the axilla were most characteristic.

In 1924, Kohlmann (6) expressed the opinion that radiological examination might help to clinch the diagnosis in cases of pulmonary infarction in which the clinical evidence was not conclusive. This

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statement reflected the general opinion held at that time, that shadows cast by infarcts were inconstant. It also revealed a lack of clearly established criteria on which a roentgenologic diagnosis could be based.

Rieder and Rosenthal (8), in 1927, made the important observation that in the presence of severe passive hyperemia in the lungs, the infarcts may fail to produce clearly defined shadows.

In 1930, Kirklin and Faust (5) made a clinical and roentgenologic study and reported 25 cases, including 17 in which the infarct had occurred postoperatively and 6 in which cardiac failure was present. They stated: "It is difficult and at times impossible to make a diagnosis of pulmonary infarction from the roentgenograms. With the clinical data, however, the nature of the shadows appearing on the film may usually be accurately determined."

As the evidence has accumulated over a period of years, the varied appearances which an infarct may assume have become more generally understood. More and more dependence has been placed on the roentgenogram to arrive at a diagnosis of a pulmonary infarct. It is probably fair to state that, in most instances of uncomplicated pulmonary infarction, it is possible to make the diagnosis of an infarct, or at least to suspect its presence, from the roentgenogram alone. The clinical data should, nevertheless, always be given due weight. In those instances in which secondary complications alter the roentgen appearance, considerable dependence must be placed on the clinical observations. This point will be discussed further under the appropriate headings.

K. S. Smith (9), in 1938, discussed the roentgen appearance of pulmonary infarcts based on his experience with 13 cases. He stated that the shadows due to infarcts of the lung fall into six groups: hazy horizontal clouding at the base; shadows suggesting pleural effusion; shadows of roughly circular form; density of one part of a lobe with cavity formation; appearance of basal collapse of a lobe; dense linear shadows.

Westermark (11), also in 1938, made the observation that in some instances of pulmonary embolus an area of avascularity can be seen corresponding to the distribution of that portion of the pulmonary artery which is occluded. He stated that the vascular markings leading to the avascular area end abruptly, and that later, if an infarct develops, an area of increased density in that location will be seen.

One year later, Jellen (4) analyzed the roentgen signs of infarcts of the lung and reported 18 cases. Among other things, he stated that the appearance of the infarct shadow may be delayed for several days following the embolic process. He also pointed out the clouding of the costophrenic sinus, the secondary formation of an abscess in some instances, and the fact that there is considerable variation in the appearance of an infarct.

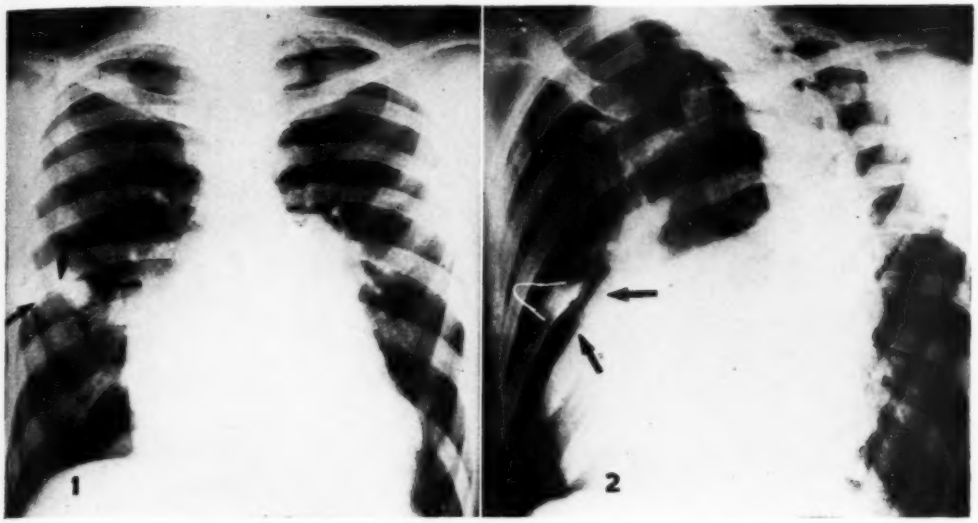
The most complete and thorough investigation thus far is that of Hampton and Castleman (3), published in 1940. Reference to their excellent work will be made at various points in this paper.

CLINICAL FINDINGS

Hemoptysis, with sudden sharp pleural pain, most frequently heralds the onset of a pulmonary infarct. A chill is almost never experienced. Dyspnea is often present. Physical examination will usually reveal râles and a change in the character of the breath sounds. If a peripheral pleural surface is involved, a friction rub may be heard. The physical findings vary with the position and age of the infarct, and are subject to modifications by the occurrence of complications such as pleural effusion and abscess.

There is moderate elevation of the temperature and of the white blood cell count, with a relative polymorphonuclear leukocytosis. The low-grade fever persists for several days and, if the patient survives, gradually returns to nomal.

Jaundice occurs occasionally. This may in some instances be due to necrosis of the infarct, but may be the result of the severe passive hyperemia which is present in



Figs. 1 and 2. These two roentgenograms demonstrate that the shape of an infarct is often dependent upon the shape of the portion of the lobe involved. A. M., a white male in his early 20's, was known to have mitral stenosis and had been observed during several episodes of myocardial failure. In the course of one of these, some months before this examination, he had had a pulmonary infarct. These roentgenograms were selected rom the group available for this patient, because they most clearly illustrate the shape of the infarct.

Fig. 1. A sharply defined oval shadow of homogeneous density (arrows) is present in the right mid-lung field, just below and adjacent to the interlobar septum. It does not reach the lateral chest wall. The cardiac silhouette is that of mitral stenosis with cardiac hypertrophy and dilatation.

Fig. 2. In the left anterior oblique projection, the shape of the shadow is that of the anterolateral tip of the right middle lobe (retouched for reproduction). There is no resemblance to the "classical" triangle, widest laterally, but instead the broadest part of the infarct is medial. An infarct in such a location must assume the shape enforced by the pleural surfaces.

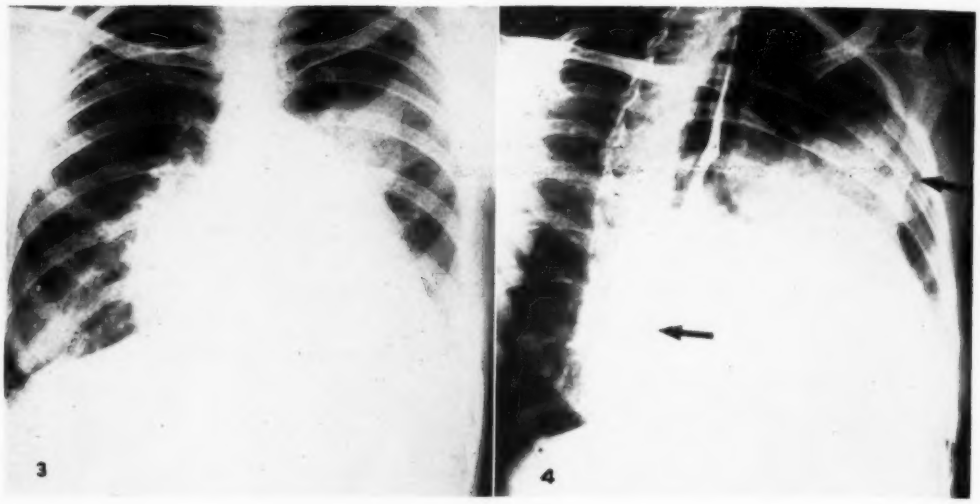
many of the patients with myocardial failure.

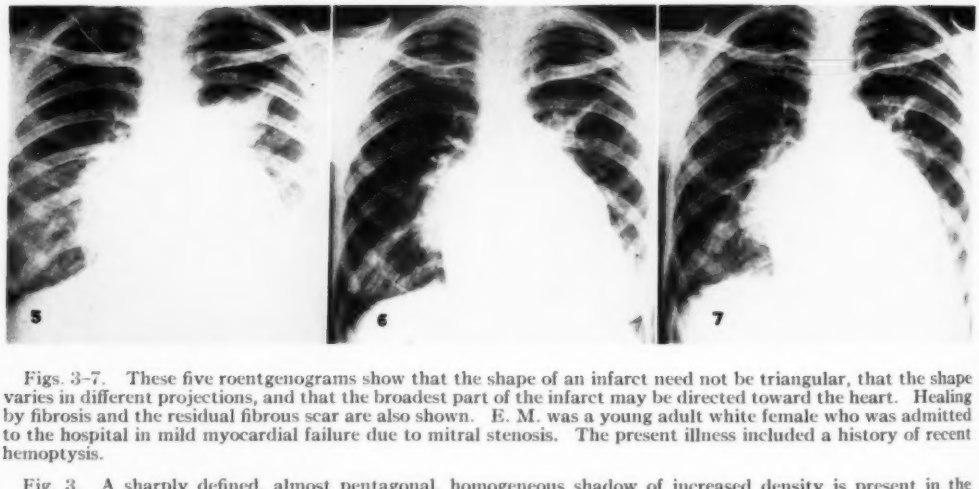
While a number of infarcts occur postoperatively, the great majority (in this series, at least) are seen in "medical" patients. Infarcts are more likely to develop in patients with cardiac disease, but a considerable number are seen in the absence of myocardial failure. In the previous study (7) 6 per cent were found to have occurred after surgery. There was evidence of heart disease in 70 per cent, and 24 per cent were in "non-cardiac medical patients." This surprisingly large number of non-cardiac medical cases is, however, quite similar to the findings of Hampton and Castleman (3). Of the 255 instances of pulmonary infarction in their series, 27 per cent were in non-cardiac medical patients. It should be noted, however, that in a patient with cardiac disease occurrence of an infarct is a much more serious event than it is in the absence of myocardial failure (7).

The occurrence of a secondary lung abscess is manifested by a higher elevation of temperature and leukocytosis and by the appearance of a foul, copious sputum, after the original symptoms have subsided. A chill may occur at this stage.

ROENTGENOLOGIC APPEARANCE

Because of the great variation in the appearance of infarcts and the frequency with which the infarct shadows are masked by various complications, repeated examinations are important. Most of these must necessarily be made at the bedside, but this should not be a deterrent, since relatively satisfactory examinations are now possible with any of the modern mobile roentgenographic units. Such units have made possible many diagnoses of pulmonary infarction in patients who were





Figs. 3–7. These five roentgenograms show that the shape of an infarct need not be triangular, that the shape varies in different projections, and that the broadest part of the infarct may be directed toward the heart. Healing by fibrosis and the residual fibrous scar are also shown. E. M. was a young adult white female who was admitted to the hospital in mild myocardial failure due to mitral stenosis. The present illness included a history of recent hemoptysis.

Fig. 3. A sharply defined, almost pentagonal, homogeneous shadow of increased density is present in the medial portion of the upper half of the left lung field. The broadest portion of this infarct shadow is *toward the heart*. There is a small pleural effusion in the left costophrenic sinus. The cardiac silhouette is that of mitral stenosis with cardiac hypertrophy and dilatation.

Fig. 4. The infarct shadow (arrows) is seen to be in the left upper lobe against the anterior pleural surface, and the borders are sharply demarcated. The widest portion is directed toward the heart. Note the posterior displacement of the esophagus (lowest arrow) and the upward displacement of the left primary bronchus by the dilated left auricle.

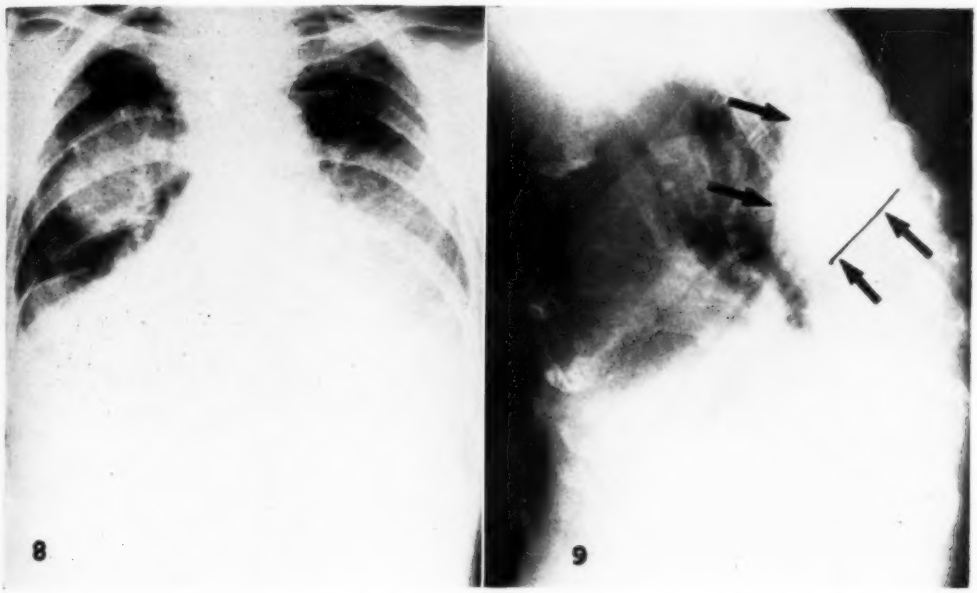
Fig. 5. One month later the infarct shadow is smaller, very sharp in outline, and still quite homogeneous. The small pleural effusion is still present. Healing is taking place.

Fig. 6. Five months after the onset the infarct shadow is seen to be much smaller, with retraction of the clear-cut borders. Strands of fibrous tissue, representing further healing, can be seen.

Fig. 7. Two and one-half years after the original examination there remains only a scar composed of strands of fibrous tissue radiating in several directions. The scar resembles that of a tuberculous lesion. If the patient had been seen at this stage for the first time, the nature of the original lesion might have been obscure. The obvious cardiac disease would then have been an important differential point in favor of a healed infarct.

too ill to be examined otherwise. The roentgenographic appearances are best discussed under separate headings, as follows:

1. *Site, Size, and Number:* All authors agree that infarcts occur most frequently in a lower lobe, especially the right. This



Figs. 8 and 9. These two roentgenograms indicate that an infarct may even appear to be round and that its appearance will vary strikingly with the projection in which it is seen. I. B., an 81-year-old colored female, was admitted to the hospital in myocardial failure due to arteriolar nephrosclerosis.

Fig. 8. This roentgenogram, made four days before death, reveals a well demarcated round shadow of increased density in the right upper lobe, surrounded on all sides by apparently normal lung tissue. There are other shadows of increased density at the right base (below the interlobar septum) and in the lower two-thirds of the left lung field. At autopsy, these latter shadows proved to be areas of confluent bronchopneumonia.

Fig. 9. The right lateral view demonstrates that the round shadow seen in the postero-anterior view is in the posterior portion of the right upper lobe, against the posterior pleura (arrows). The shape in this projection is roughly that of a truncated triangle (retouched for reproduction). The value of additional views to demonstrate the true shape is thus evident. The roentgen diagnosis of an infarct in the right upper lobe was confirmed at the postmortem examination.

was also observed in the previous study (7). When an upper lobe is involved, infarcts will almost always be found in a lower lobe as well.

Infarcts may vary from less than 1 cm. in diameter to an area occupying an entire lobe. Most frequently they measure from 2 to 6 cm. Once developed, the infarct will vary but little in size (except for healing), but complications such as pneumonia, abscess, and pleural effusion may cause an apparent increase in the extent of the shadow.

Infarcts are more frequently multiple than single, but only rarely are more than two or three seen on the roentgenogram. Instances of as many as 14 visible infarcts have been reported (3), but are exceptional.

2. *Shape:* There is no characteristic shape of an infarct shadow. The earlier idea that the shadow as seen on the roentgenogram was triangular, with the base laterad, was drawn from the theoretical concept that infarcts were conical in shape. This has been disproved by Hampton and Castleman (3). By the nature of the pathologic process an infarct must be so situated that it borders on at least one pleural surface. It follows that the outlines of an infarct shadow are subject to the modifications imposed by the contour of the portion of the lobe involved (Figs. 1 and 2). It can readily be seen that an infarct located at a point in the parenchyma where two or more pleural surfaces meet must assume the shape of that portion of the lobe. The variations in the shape of the shadow of a pulmonary infarct are demonstrated in Figs. 1 to 4, 8 and 9, 11, 13, and 18.

It is possible for an infarct to have a

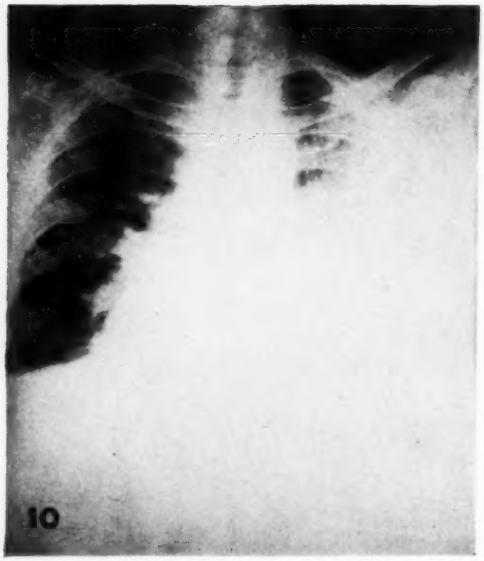


Fig. 10. In this case a pleural effusion completely hides an infarct. T. B., a 56-year-old white male, was admitted to the hospital in myocardial failure. There was a recent history of hemoptysis and fever. The roentgenogram shows a large pleural effusion in the left half of the chest. Following the removal of the fluid (which was blood-tinged and foul), an abscess cavity within an area of consolidation was demonstrated. This was treated surgically, and recovery ensued. Six months later the patient died in cardiac failure and postmortem examination revealed a stellate scar in the left upper lobe, with organized thrombi in the corresponding branches of the pulmonary artery. Thus, on the roentgenogram, a pleural effusion completely obscured a pulmonary infarct. However, because the clinical data were quite definite, the correct diagnosis was made.

conical shape and to present a roughly triangular appearance on the roentgenogram (with the apex truncated and the base peripheral) but, if so, such a triangular appearance can be present in only one projection (Figs. 8 and 9). Unless the base of the infarct is against the lateral chest wall, the roughly triangular shape will not be apparent in the usual postero-anterior projection and will be seen, if at all, in an oblique or lateral view.

Variations in the shape will also be encountered as the result of superimposition of more than one infarct shadow (3). It may be possible to separate such shadows by additional views. Infarcts located in the costophrenic angle may be triangular, but because of the shape of that part of the lung, the widest portion is toward the heart.

In large infarcts, as the result of the collapse of the large numbers of alveoli, the volume of the lung is somewhat reduced, occasionally to a degree simulating collapse of a lobe (3). The diaphragm may be elevated, especially if the peripheral pleura is involved.

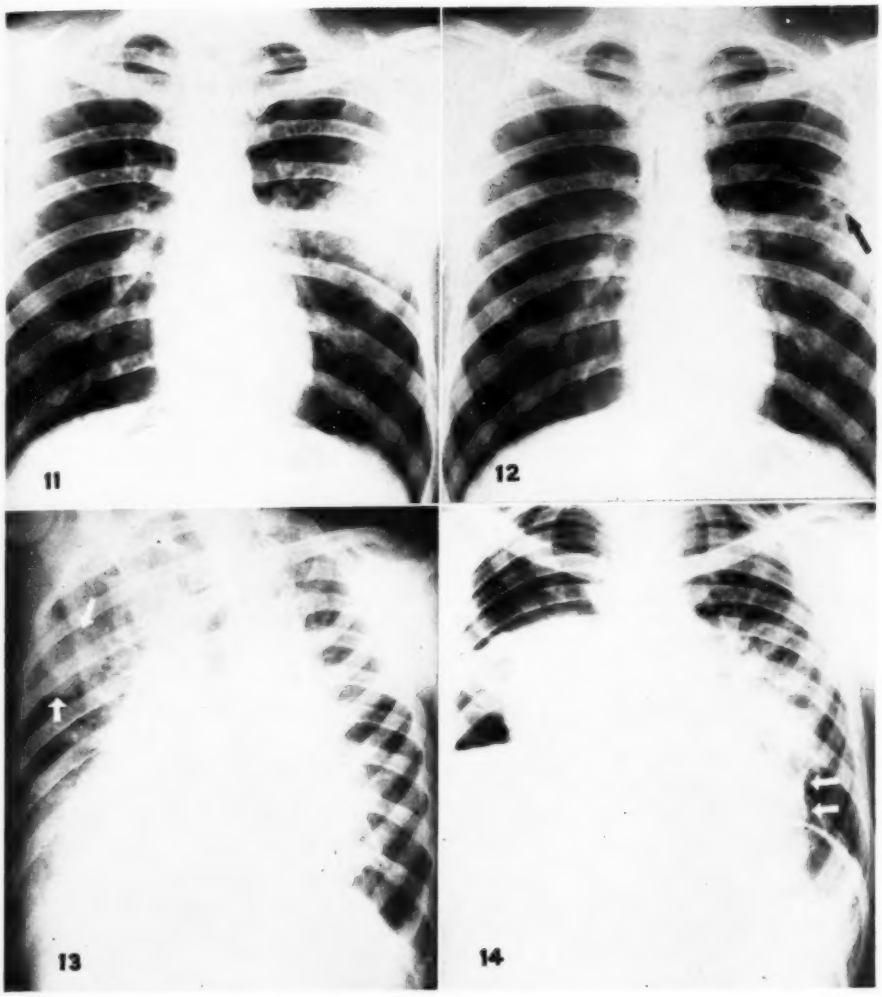
The borders of an infarct shadow are usually sharply demarcated (Figs. 2, 3, 11, and 13). This is especially true of the pleural border or borders, but the parenchymal borders are also relatively clear cut. At the cardiac side (point of occlusion of the artery) the shadow may be slightly fuzzy.

3. *Density:* The shadow of a pulmonary infarct is almost always homogeneous after the first few hours, and is of moderate density in relation to its diameters (Figs. 3, 8, 11, and 13). Since the lesion is due to occlusion of a blood vessel, the alveolar collapse and necrosis which ensue are quite uniform. The formation of a lung abscess and the process of healing will alter this appearance.

4. *Complications:* The presence of various complications may obscure the roentgenologic appearance, sometimes quite early in the course of the infarction. As previously stated, in such instances it may then be impossible to make the correct diagnosis without help from the clinical data. Roentgen examination should, therefore, be made as soon as possible after the onset of symptoms.

Secondary *bronchopneumonia* may begin within a few days after the onset of an infarct. In such instances, the non-pleural borders of the infarct lose their relatively sharp outline, assume a feathery appearance, and fade into the surrounding lung parenchyma (Fig. 15).

A *pleural effusion* is a distressingly frequent complication. At times an effusion is present before the infarction occurs; again, it develops soon afterward. In some cases the involvement of the peripheral pleura by the infarct is responsible for the effusion; more often, myocardial



Figs. 11–14. These two cases and that shown in Fig. 15 demonstrate the effect of secondary infection with abscess formation and pulmonopleural fistula on the roentgen appearance of an infarct. In all of these cases the correct diagnosis would have been difficult, if not impossible, without the aid of the history, had the patients been seen for the first time in the later stages of the illness. With the serial films which were available in each case, however, the roentgen diagnosis was not difficult.

Figs. 11 and 12. T. A., a 20-year-old white male, had hemoptysis and pain in the left side of the chest four days after a submucous resection of the nasal septum. On a roentgenogram taken one day later (Fig. 11) there is a homogeneous shadow of increased density in the upper portion of the left lung field, laterally. It is roughly triangular in this projection, the lower border is sharp, the upper border fairly well demarcated.

Four weeks later an abscess cavity about 2 cm. in diameter is visible within the shadow of the infarct (Fig. 12). The infarct shadow is no longer homogeneous, but is the seat of fibrosis and retraction indicating some healing.

Figs. 13 and 14. F. B., a 28-year-old white male with mitral stenosis, in moderately severe myocardial failure, complained of pain in the right side of his chest and of repeated small hemoptyses. In the left anterior oblique roentgenogram made two days after the first hemoptysis (Fig. 13), there is a clear-cut shadow of even density in the anterior portion of the right upper lobe, adjacent to the anterior and the interlobar pleura (arrows), representing an infarct. In the postero-anterior view, the shadow was ovoid.

Five days later an abscess cavity was demonstrated within the infarct, and the next day a pneumothorax, containing a fluid level was demonstrable (Fig. 14). The right lung is collapsed and adhesions to the chest wall are visible. The sharp border of a fresh infarct in the left lower lobe, just lateral to the cardiac border, is also seen (arrows). At postmortem examination, the same day, the roentgen diagnosis of an infarct in the right upper lobe with abscess formation, pulmonopleural fistula, and hydropneumothorax was confirmed. The pleural fluid was foul.

Fig. 15. This figure, like Figs. 11–14, shows the effect of secondary infection with abscess formation on the roentgen picture of an infarct. W. H., a 51-year-old white male, had myocardial failure due to coronary sclerosis. There is an ovoid area of increased density in the lower anterolateral portion of the right upper lobe (arrows). The borders of this shadow are in part clear cut, and in part indistinct (due to secondary bronchopneumonia). Within the shadow of the infarct there is an abscess cavity. At the right base there is a small pleural effusion, and at the left base a pneumothorax pocket (following thoracentesis). Serial films in this case demonstrated the appearance of the infarct, then the abscess, and finally the healing of both. This type of dense shadow, against a pleural surface and containing an abscess, should suggest the correct diagnosis on the roentgen evidence alone, but the clinical data are most helpful.

failure is the cause, but in many instances both factors are active. A unilateral effusion, however, appearing after an infarct on the same side and containing bile pigments, is probably due to the infarct. (Bile pigments are not seen in a pure transudate.) In the absence of myocardial failure, the infarct must be considered as the cause of the effusion.

In any case, the development of a pleural effusion complicates the roentgen diagnosis of an infarct by obscuring the infarct shadow (Fig. 10). This is one type of case in which the history is quite important. The frequent relationship between effusion and infarction should place the roentgenologist on guard.

The secondary formation of a *lung abscess* within an infarct is an unfortunate but not uncommon sequel. This has long been recognized as a frequent occurrence in instances of infected emboli (as in septicemia), but it may occur even though the infarct was originally bland, and in the presence of repeatedly negative blood cultures. The route of infection is probably *via* the bronchi. Seventeen such cases were seen in the material studied and have been previously reported (1). The clinical course has been described.

Roentgenologically there is observed an area of diminished density within the shadow of the infarct (Figs. 12 and 15). The non-pleural borders are frequently fuzzy because of the presence of some pneumonia. If the patient is seen early in the course of his illness and followed throughout, the sequence of events is quite characteristic. If he is seen for the first time with the cavity already present, the diagnosis of an underlying infarct may easily be overlooked, because of the resemblance to any lung abscess. If, however, an area of diminished density is seen within a fairly well demarcated shadow of increased density, situated against a pleural surface, the diagnosis should certainly be suggested from the roentgenogram alone (Fig. 15). Again, in this type of case, due weight must be given to the history.

Following the occurrence of an abscess, but especially if the peripheral pleura is involved, a *pulmonopleural fistula* may develop and an empyema result. This sequence of events occurred 7 times among the 17 cases mentioned above (1) (Figs. 13 and 14).

5. *Healing:* Many small infarcts heal. This number is probably greater than is generally realized, since the correct diagnosis has often been missed. Healing of an infarct does not occur by resolution. Once an infarct has completely developed (as opposed to the incomplete infarction described by Hampton and Castleman), the necrotic tissue will be replaced by fibrous tissue, if healing occurs at all. This will result in a scar, visible on the roentgenogram (Figs. 3–7, 18 and 19). The finding of such scars, which may be stellate or linear, especially in the lower lobes, should

lead to the consideration that they represent old healed infarcts. They must be differentiated from disks of atelectasis and from interlobar pleurisy. The differential diagnosis is discussed under the appropriate heading.

DIFFERENTIAL DIAGNOSIS

The infarct shadow must be differentiated from pneumonia of various types, neoplasm, passive hyperemia, pleural effusion, cysts, and atelectasis.

1. *Lobar Pneumonia:* Lobar pneumonia is usually manifested by a homogeneous shadow of increased density, which may present a sharp border if it reaches an interlobar surface. Except for the frequent occurrence of a chill, the clinical history approximates that of an infarct. The amount of lung parenchyma involved is much greater than that of the average infarct. If the patient is seen early, a small area of infiltration may be observed to spread rapidly in the succeeding twenty-four to forty-eight hours, even though the patient may be responding favorably to chemotherapy. This very rapid and marked increase in size of a homogeneous shadow is not seen in an infarct. Shadows due to lobar pneumonia (as seen in the postero-anterior view) are usually more dense than shadows of equal size due to an infarct, since in most instances of lobar pneumonia a greater depth and therefore greater volume of lung parenchyma is involved. It is rare for an infarct to involve an entire lobe, while a lobar pneumonia does so very frequently.

Furthermore, serial roentgenograms will demonstrate that, in many instances, the shadow of a lobar pneumonia begins medially and extends across the lung field (Fig. 16), whereas an infarct is always in contact with a pleural surface from the beginning. When an infarct borders on the lateral pleural surface, a clear area is almost always present medially (Figs. 11 and 15).

2. *Bronchopneumonia:* The shadows caused by bronchopneumonia are usually basilar, frequently bilateral, patchy and mottled. The density of these multiple small areas is uneven, and the edges are fuzzy and fade into the surrounding lung. Most of the areas are not in contact with a pleural surface. On the other hand, the infarct shadow has more clear-cut margins and a more even density; it is always against a pleural surface, and only occasionally are more than two or three infarct shadows visible. Shadows due to bronchopneumonia will show more variation from day to day.

3. *Atypical Pneumonia:* This recently described disease of unknown etiology has little resemblance to pulmonary infarction from a clinical standpoint. The patients are usually not critically ill, hemoptysis is rare, and there is almost always a history of an immediately preceding upper respiratory infection.

Roentgenologically, atypical pneumonia resembles bronchopneumonia, or what some roentgenologists have called "interstitial pneumonia." The same multiple small patches of uneven density, occasionally streaked in appearance, are seen. The margins are hazy, and the patches are surrounded by normal lung. The areas of infiltration occur most frequently in the lower lobes but may be seen anywhere in the lung fields. Not infrequently they are subapical in position.

There is a greater tendency to "migration" than in bronchopneumonia, *i.e.*, resolution occurs in one area while fresh patches appear elsewhere. The differential diagnosis is the same as for bronchopneumonia.

4. *Neoplasms:* Since one of the effects of occlusion of a branch of the pulmonary artery is collapse of alveoli in the area of the infarct, a large infarct may cause sufficient diminution in the volume of a lobe to simulate the collapse produced by an endobronchial neoplasm. Hampton and Castleman (3) have pointed out that the use of an over-penetrated roentgenogram will usually fail to reveal areas of normal lung density within the shadow of the collapsed lobe due to endobronchial tumor, whereas a lobe occupied by several infarcts is more

likely to be of uneven density. They further point out that a collapsed right middle lobe can be differentiated by its shape and by its retraction downward and medially, whereas an infarct shadow in this location does not reach the lung root.

Metastatic tumor nodules may be differentiated by the fact that they are rounded, regardless of the projection in which they are seen. They are often multiple and are rarely situated against a pleural surface. It is rare for an infarct shadow to be perfectly round, and still rarer for more than one to have this appearance. Even when an infarct shadow is relatively round in the postero-anterior view, its shape will vary in other projections (Figs. 8 and 9).

5. *Passive Hyperemia:* Cases of myocardial failure with considerable passive hyperemia in the lung fields will present a rather dense bilateral shadow extending laterally from the hili and fading out toward the periphery. This may cause the outlines of an infarct to become indistinct and to resemble a patch of bronchopneumonia. The shadow resulting from the passive hyperemia, together with the large cardiac shadow, will hide a large portion of the lung fields. If an infarct happens to be situated anteriorly or posteriorly and close to the mediastinal border of the lung, it may be completely hidden (Fig. 21). The problem here is not so much one of differentiation as remembering that this may occur, so that if the clinical story suggests an infarct, roentgenograms in additional projections may be obtained.

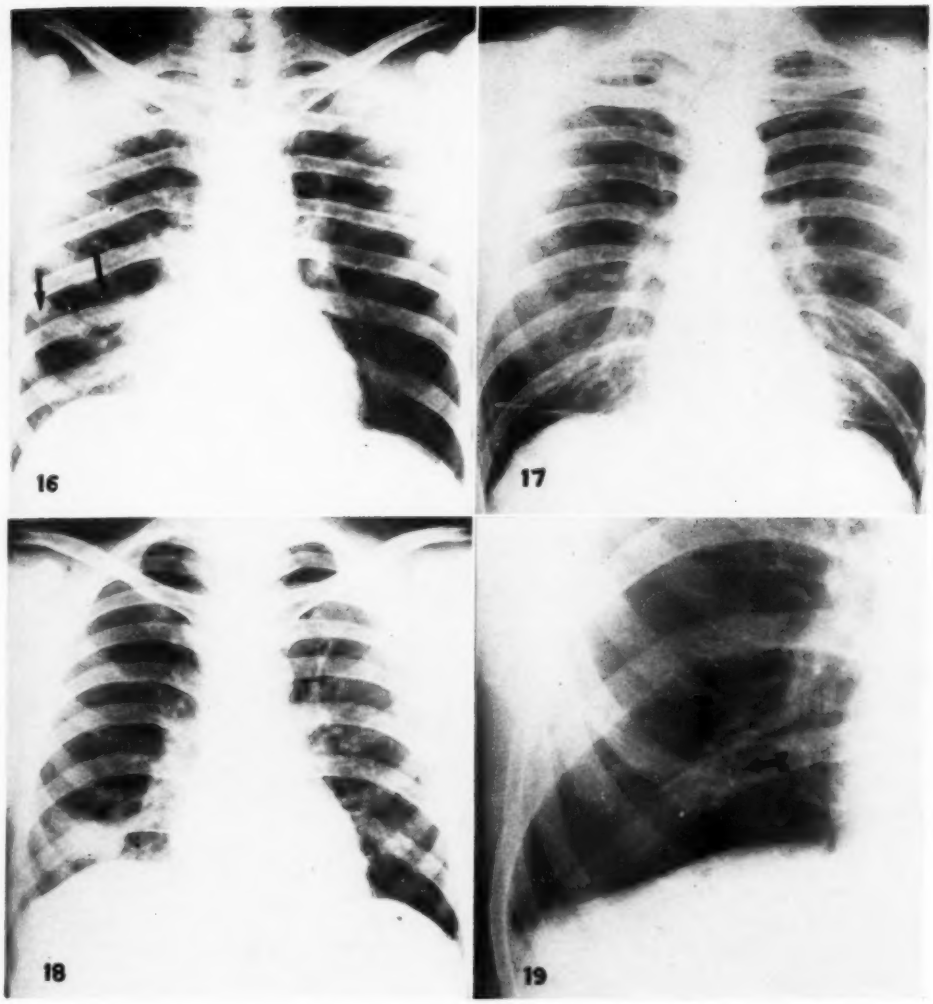
6. *Pleural Effusion:* Differentiation from a pleural effusion, either peripheral or interlobar, is one of the most troublesome problems. In the free pleural effusion, a dense homogeneous shadow is present in the lower lateral portion of the lung field with a smoothly curved upper border, convex downward and higher laterally. An infarct which occurs in the lower lateral portion of a lower lobe may be confused with this, but the upper border of the infarct is more likely to be straight (although not necessarily horizontal) or "humped," *i.e.*, convex upward. The co-existence of the two entities is frequent. The presence of a large obvious pleural effusion will often mask an underlying infarct (Fig. 10).

An infarct with one border against the interlobar fissure on the right may be confused with an interlobar effusion. An infarct, however, will not be elliptical, and an interlobar effusion almost always is. The interlobar effusion often extends from the hilus to the lateral chest wall, or at least pleural thickening can be seen to reach these points even if the effusion is confined to the central portion of the fissure. It would be practically impossible for an infarct in the lower anterior portion of the right upper lobe to extend entirely across the lung field and still be narrow enough to simulate an interlobar effusion. Furthermore, if the infarct is in the middle lobe, it cannot reach the lateral chest wall.

7. *Cysts:* Cysts, when they are air-containing or when a fluid level is present, offer no difficulty in differential diagnosis. When they are completely filled with fluid and only one or two are present, they may present a problem. A fluid level, however, if not present originally, will usually be detected on one of a series of examinations, and cysts are rarely situated against a pleural surface. Echinococcus cysts may have a calcified border. The rounded contour of a cyst (in all projections) is an important differential point. Differentiation is usually not difficult.

8. *Plaques of Atelectasis:* In the very early stages of an infarct, only a few horizontal streaks of increased density may be visible (Fig. 20). In a matter of a few hours the infarct shadow will assume the more characteristic features already described. Therefore, differentiation from atelectatic disks is best made by repeated examinations, since the latter will change in appearance and often disappear.

More confusing, and more frequently encountered, is the problem of differentiating between the scar of a healed infarct, such atelectatic disks, and interlobar pleurisy. Fleischner, Hampton, and Castleman (2) point out that thickening of the



Figs. 16–19. This group of three cases and that shown in Fig. 20 illustrate the problem of the differential diagnosis between the scars of healed infarcts, interlobar pleurisy, and plates of atelectasis. The occasional simulation of such atelectatic areas by an early infarct is also shown.

Fig. 16. E. W., a 34-year-old white male, was acutely ill. The interlobar pleura (arrows) is seen as a sharp, narrow line extending completely across the right lung field. It is in a slightly lower position than is usually seen, since this is a bedside examination, but it is clearly identifiable as an interlobar fissure. In the medial portion of the right lower lung field there is a dense homogeneous shadow with a fuzzy lateral border, representing a lobar pneumonia in the right middle lobe. The hazy lateral border distinguishes this shadow from that of an infarct. Within twenty-four hours the entire right middle lobe was consolidated.

Fig. 17. S. B. In the lower portions of both lung fields there are horizontal streaks of increased density, single on the left and double on the right. These streaks extend almost completely across the lung fields, are approximately parallel, and represent disk-like plates of atelectasis.

Figs. 18 and 19. F. Y. In Fig. 18 an oval shadow of increased density is visible just above the right leaf of the diaphragm. This represents a pulmonary infarct caused by an embolus from a thrombophlebitis in the leg. The borders are sharply defined and the shadow is of even density. In a coned-down view of the right base made two years later (Fig. 19) a stellate scar of fibrous tissue is seen. This is to be differentiated from a plaque of atelectasis by the fact that the strands are multiple but not parallel, are localized to a small area, and do not extend across the lung field in a horizontal direction. (See also Figs. 3–7 for fibrous scar formation.)

Fig. 20. D. W. Occasionally an early infarct will simulate plaques of atelectasis. In this case of septicemia there are present only a few strands of increased density just above the right leaf of the diaphragm. They are multiple, and roughly parallel, but are short. At postmortem examination, within a few hours, small recent infarcts were found in this area. Had there been an opportunity for another examination, the infarcts probably could have been demonstrated in a more characteristic form.

interlobar pleura can be recognized by the fact that the anatomical location of the shadow corresponds to that of a normal fissure. Other evidences of pleurisy may be present. Infarct scars may run in any direction, but always reach a pleural surface and often have a nodular thickening at that point. Infarct scars are usually shorter than atelectatic plates; they may be multiple and in different planes (Figs. 7 and 19). The atelectatic shadows are long, horizontal, and when multiple are almost parallel (Fig. 17). They vary in appearance from day to day, whereas infarct scars do not change. The activity of the diaphragm is limited in the presence of atelectasis, but is not impaired in healed infarcts. Both are most frequent in the lower lobes, but infarct scars are not uncommon in upper lobes, whereas atelectatic plates are rarely seen there.

SUMMARY

The frequency with which the diagnosis of a pulmonary infarct is missed, both clinically and roentgenologically, has been noted. The clinical syndrome has been briefly reviewed. The roentgenologic observations and the differential diagnosis have been discussed in detail. While the roentgen appearance of a pulmonary infarct may vary widely, the features most commonly present are as follows:

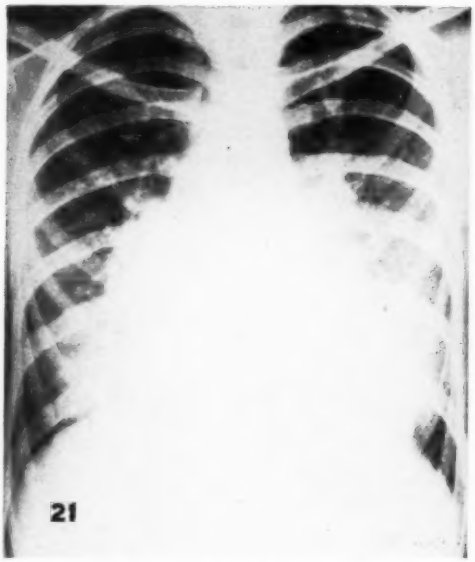


Fig. 21. This case demonstrates the fact that an infarct may be hidden by a large heart and passive hyperemia. A. J., a 16-year-old female, was admitted to the hospital in myocardial failure due to rheumatic pancarditis. The cardiac shadow is greatly enlarged and the presence of passive hyperemia is indicated by the bilateral fan-shaped shadow extending from both hili. There is no shadow suggesting the presence of an infarct. At postmortem examination, within a few hours, a large organizing infarct was present in the medial portion of the right lower lobe. In spite of its size it was hidden by the large heart and passive hyperemia. Thus it should be remembered that if the clinical story is that of an infarct, additional views should be made to visualize otherwise hidden portions of the lungs.

1. The shadow of increased density is homogeneous.
2. The margins of the shadow are sharply demarcated.
3. The infarct shadow is more often present in a lower lobe.
4. It borders on one or more pleural surfaces (peripheral or interlobar).
5. The size and shape vary widely. The shape is often dependent on the shape

of that portion of the lobe which is the site of the infarct. Occasionally the shape is that of a truncated triangle.

6. Infarcts are usually multiple, but only rarely are more than two or three large enough and so situated as to be visible roentgenologically.

Pleural effusion, bronchopneumonia, and lung abscess (sometimes with pulmonopleural fistula and empyema) may follow the development of a pulmonary infarct and obscure the roentgenologic appearance. In such cases the underlying infarct may be difficult and even impossible to recognize roentgenologically without the aid of a carefully taken history.

The shadow of the pulmonary infarct may be confused with and must be differentiated from pneumonia (lobar, bronchial, and atypical), neoplasm (primary and secondary), pleural effusion (free and interlobar), atelectasis (massive and platelike), chronic passive hyperemia, and cysts. Criteria for the differential diagnosis have been presented.

Infarcts, especially small ones, may heal. Healing takes place by fibrosis and not by resolution, once an infarct has completely developed. In such instances stellate or linear scars may be visible in the lung fields. These must be differentiated from plates of atelectasis.

CONCLUSION

Roentgenographically a pulmonary infarct may mimic almost any other lung lesion. With a careful analysis of the roentgen appearance, however, and with consideration of the clinical data, the nature of the shadows should be accurately determined in a large proportion of cases. As has been so often stated, the most frequent cause of error in the diagnosis of any disease lies in the failure to consider it as a possibility.

NOTE: Figures 13, 14, 18, 20, and 21 are reproduced by courtesy of *Archives of Internal Medicine*.

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The Roentgen Appearance of Lobar and Segmental Collapse of the Lung

IV. Collapse of the Lower Lobes[1]

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DIAGNOSIS of collapse of individual lobes or segments of lobes requires criteria additional to those which are necessary in collapse of an entire lung or the major part thereof (7, III). Besides the well recognized signs, therefore, the roentgenologist must look for any change from normal in the position of the hili and in the position and contour of the septa, and for rearrangement of the vascular shadows.

Collapse of a lower lobe occurs rather frequently, but it is often overlooked or misinterpreted because the shadow of increased density does not stand out as clearly as in other pathologic processes, such as consolidation. The shadow may be small and so located that it is hidden by overlying structures, as by the mediastinum and diaphragm on the postero-anterior roentgenogram and the spine on the lateral roentgenogram. Roentgenologically, the two lower lobes are essentially alike both in structure and position, so that the appearance of collapse of the whole lobe or of its segments is similar on the two sides. One important difference is that disease processes in the left lower lobe are often obscured by the heart shadow, but with the technic previously described (7, I) this difficulty is overcome. In the 600 cases studied, the left lower lobe was more frequently involved than the right, the left being collapsed in 262 cases and the right in 156.

In the majority of instances, it is possible, with satisfactory technic, to demonstrate the location of the septa which bound the individual lobes (7, II). Roughly, each major septum runs from the level of the fifth thoracic vertebra posteriorly to a point near the anterior portion of the diaphragm. The pulmonary structures seen posterior to the greater septa represent the lower lobes. On the right side a fair portion of the lower lobe is behind both the upper and middle lobes, while on the left, the upper lobe and its lingular portion lie in front of the lower lobe. The remainder of the lower lobes lies below the shadow of the dome of the diaphragm. It is evident, therefore, that in the postero-anterior projection essentially the entire lower lobe on each side is either superimposed on shadows of the adjoining lobes or is obscured by the diaphragm.

Frequently the first evidence to suggest a decrease in size of a lower lobe is the demonstration on the lateral roentgenogram of change in the normal position of a major septum, which appears to lie further posteriorly than usual. Shortly after this becomes evident, it is observed that a large number of the pulmonary markings of the lower lobe tend to radiate from the hilus downward and posteriorly just anterior to the spine, where they fan out anteriorly and posteriorly in gentle curves. As the collapse progresses, it soon becomes apparent that there is actual crowding together of the lung markings and that the collapsing lung occupies less and less space. At this stage the closely grouped pulmonary markings stand out as a definite shadow of increased density in contrast to the adjacent uninvolved lung. The middle lobe, or the lingula, and the adjoining portions of the upper lobes will then appear emphysematous, filling the space formerly occupied by the collapsed lower lobe, which has now moved posteriorly and medially.

Collapse of an entire lower lobe casts a shadow of increased density of rather char-

[1] From the Department of Radiology, Massachusetts General Hospital, Boston, Mass. One of a series of papers accepted for publication in October 1944.

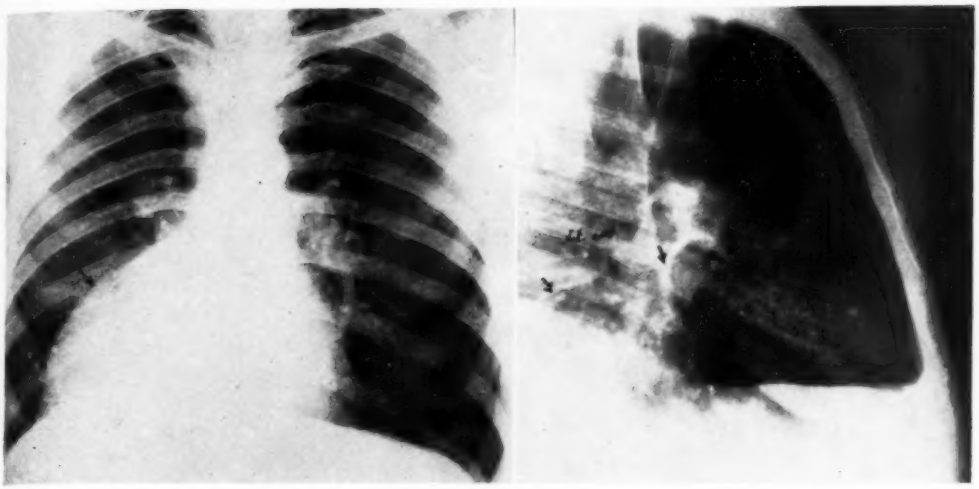

Fig. 1. Collapse of right lower lobe due to epidermoid carcinoma, grade 3.

1. The shadow of increased density, due to the tumor and the collapsed right lower lobe, obscures the right border of the heart and the medial and posterior half of the right diaphragm.
2. The right upper and middle lobes show emphysema. It is particularly marked in the middle lobe.
3. The minor septum extends to the posterior chest wall. The major septum on the left is in normal position.
4. The right hilus is slightly low in position.
5. The mediastinum is displaced to the right.

(A small amount of retained iodized oil is seen in the region of the middle lobe.)
Pneumonectomy was performed.

acteristic shape and position in the chest (4). In many cases, it has been possible to follow the actual development of this shadow from the first interference with aeration of the lobe, and consequent failure to change from the expiratory to the inspiratory phase, to the final stage, when the lobe is seen as a dense mass of atelectatic lung lying posteriorly and medially in the pleural cavity. In acute obstruction, this process will take place much more rapidly than in chronic collapse. The shadow appears as a pyramidal area of increased density with its apex at the inferior portion of the hilus and its base posteromedially on the diaphragm. It usually obscures the immediately underlying diaphragm and adjacent postero-inferior thoracic wall. (It should be noted that the lobe can be considerably decreased in size without a great amount of atelectasis being visible; the comparative difference in density of this shadow of collapsed lung and the adjoining lung is slight.) If the lesion is a chronic one, in which secondary fibrosis is set up, as in bronchiectasis, the patient may go for years in a stationary phase, showing, on roentgen examination of the chest, findings such as those described above (Figs. 1 and 2). In bronchiectasis, the shadow cast is often described as a triangular shadow of increased density (3). As collapse of the lobe becomes marked, the space formerly occupied by it is filled with the adjacent emphysematous lung. If the cause of interference with aeration of a lower lobe is acute and is relieved, the process will reverse itself and the shadow will disappear.

Not only may the diaphragm be elevated and show diminished motion but, in the earlier phase of collapse, a large portion of it may be obscured by the decreased aeration in the involved lower lobe; if the atelectasis progresses to become complete, the diaphragm is increasingly obscured, with eventual loss of definition of the portion in contact with the collapsed lobe. In chronic cases, the collapsed lower lobe occasionally moves so far posteriorly

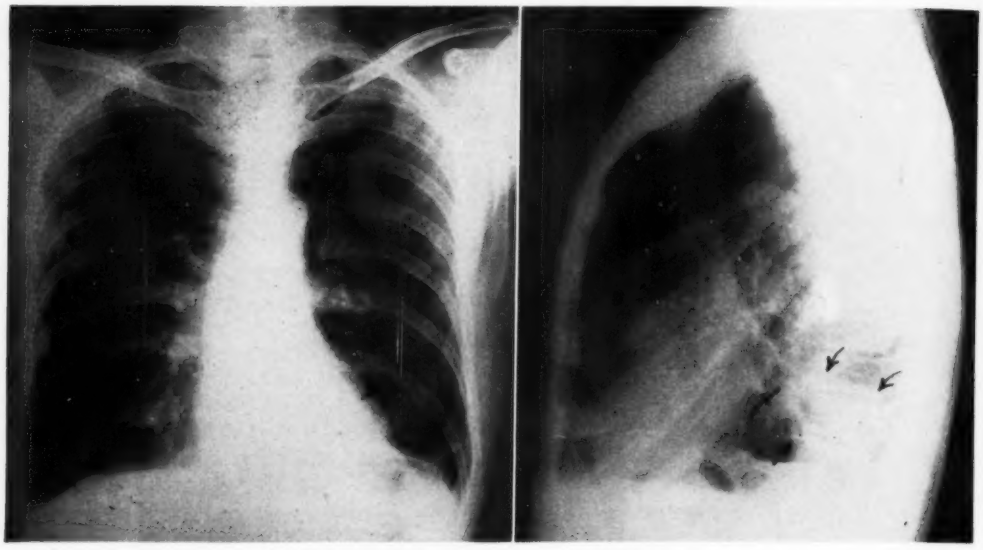

Fig. 2. Collapse of the left lower lobe due to adenoma of the left lower lobe bronchus.

1. The edge of the shadow of increased density is seen through the heart shadow (arrows).
2. The left hilus is low in position.
3. All of the visible air on the left side is in the upper lobe.
4. There is elevation of the diaphragm.
5. In the lateral view, the arrows indicate the space occupied by the shadow of increased density. Note the loss of definition of the posterior portion of the diaphragm and the adjacent chest wall.

A left lower lobectomy was performed.

and medially, and occupies such a relatively small area in the chest, that the dome of the diaphragm will be projected above the obscured portion, thus giving the impression, in both postero-anterior and lateral roentgenograms, that the entire diaphragm is sharply outlined. When this occurs, it becomes necessary to study the entire surface of the diaphragm, preferably first by fluoroscopy, in order to demonstrate that a portion is obscured by atelectatic lung above. In many instances, particularly in chronic collapse of a lower lobe, it may not be possible to demonstrate elevation of the diaphragm, shift or displacement of the mediastinum, or narrowing of the rib spaces.

As a lower lobe decreases in size, the hilus tends to move into a lower plane, and its inferior portion may be assimilated in the shadow of increased density. This perhaps explains why the hilus at times appears to become smaller. At other times the displacement of the hilus may carry it behind the shadows of superimposed structures, again creating an impression of decrease in size. In general, the depression of the hilus varies with the degree of collapse, but in certain instances, as in the presence of a fairly large tumor or of collapse in a basilar segment with marked emphysema in the dorsal division, the depression, if present, may be only slight. Change in the position of the hilus may be readily observed in patients whose collapse is of a temporary nature, for with complete re-expansion of the lobe the hilus resumes its normal position.

Any marked change in the position or contour of the septa may be of importance in determining the area of the lung in which collapse is present and the degree to which it has progressed. This is particularly true in segmental collapse. The septa have been previously noted as being normally straight or gently curved lines (7, 11). When an entire lower lobe is collapsed, the major septum retains its usual contour but

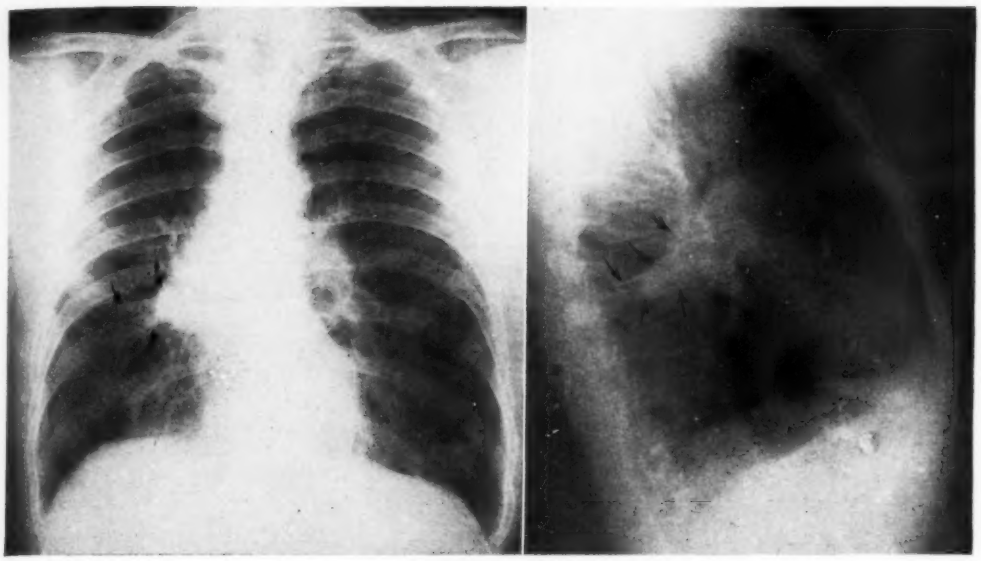

Fig. 3A. Collapse of the dorsal division of the right lower lobe due to epidermoid carcinoma. The sharply defined shadow of increased density is outlined in the postero-anterior and lateral roentgenograms and is characteristic in position and shape. See also Fig. 3B.

is displaced posteriorly (8). In collapse of the right lower lobe, the minor septum may extend to the posterior chest wall, since the middle lobe may occupy all of the area directly adjacent to the diaphragm except the small part occupied by the collapsed lower lobe. Furthermore, the minor septum is often, though not always, displaced downward from its normal position.

Rearrangement of the uninvolved lung adjoining the collapsed lower lobe explains the development of compensatory emphysema which is always present and usually appears as a variation in brightness in the lung fields. This apparent brightness is caused by decrease in the number of vascular markings and the other shadows per unit area. The normal vascular shadows have been separated (and therefore lessened in number) by the increased size of the air-containing spaces. Comparison of the emphysematous lung with the opposite normal lung will show that it contains considerably fewer opaque structures.

Fig. 3B. Same case as Fig. 3A: left anterior oblique bronchogram. The shadow of density is outlined by arrows. There is no filling of the dorsal division bronchus. Right pneumonectomy was performed.

It should be stressed that, because of the anatomic arrangement of the bronchi, a lesion which completely occludes the bronchus to the right lower lobe may also oc-

Fig. 4. Atelectasis of the posterolateral segment of the right lower lobe with broncholiths and bronchiectatic abscess. Arrows outline the shadow of increased density. The figure 1 indicates one of the broncholiths. A right lower lobectomy was performed.

clude that of the right middle lobe, so that simultaneous collapse of the right middle and lower lobes is of frequent occurrence. Stenosis of the left lower bronchus, on the contrary, affects the left lower lobe only, since the bronchus to the lingula, the correlative of the right middle lobe, arises from the left upper lobe bronchus. In bronchiectasis, however, the lingula is often found to be involved in combination with the left lower lobe. In a few cases, a collapsed lower lobe was seen to lie very much farther anteriorly than has been described, and this was interpreted as representing congenital collapse. This supposition has not, however, been proved.

For practical purposes the lower lobe can be divided into the posterior upper portion, or the so-called dorsal division, and the basal portion. The bronchus to the dorsal division of the lower lobe arises from the main bronchus at the level of the middle lobe orifice on the right and just distal to the upper lobe orifice on the left. The basal portion, composing the remainder of the lower lobe, occupies the space posterior to the middle lobe on the right and the lingula on the left.

Collapse confined to the dorsal division of the lower lobe (Fig. 3) is encountered somewhat less frequently than collapse of the basal portion or collapse of the entire lower lobe. It can be recognized without great difficulty, since it produces a rather characteristic shadow with a definite localization and a marked change in the appearance of the septa. The shadow of increased density, as in collapse of the entire lower lobe, is due to the drawing together of normal structures. A very small area of atelectasis may result, which in the postero-anterior projection is seen to have a rather sharp upper margin and to lie slightly below the hilus. It is often misinterpreted as an area of segmental collapse of the middle lobe. The lateral view, however, shows it to be located posteriorly in the chest, occupying part of the space ordinarily filled by the fully expanded dorsal division. In both projections, there is a tendency for the shadow to assume a triangular shape. In the postero-anterior roentgenogram the base lies against the mediastinum and the apex extends laterally, whereas in the lateral view the apex is directed posteriorly and the base lies

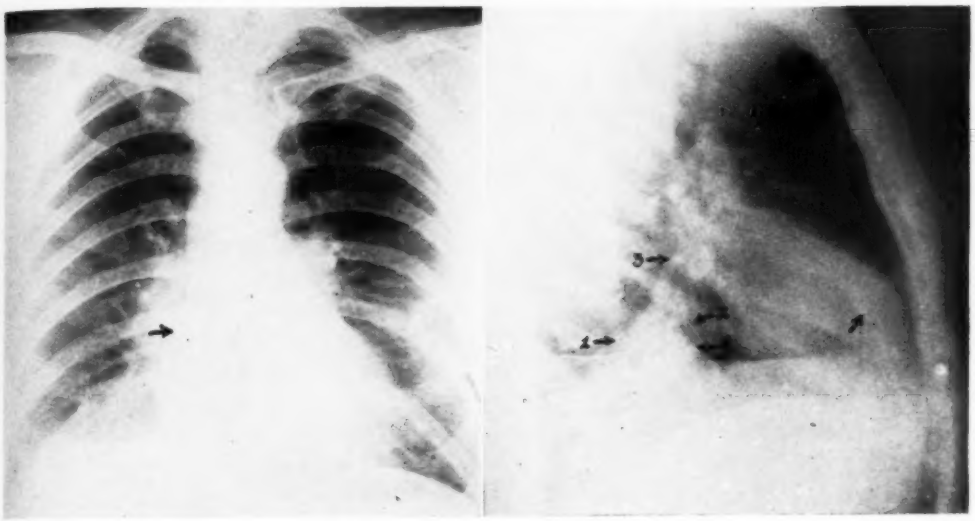

Fig. 5. Collapse of the anterior segments of the right lower lobe, probably due to a calcified node.
Postero-anterior roentgenogram: 1. The right border of the heart is poorly defined. 2. The right hilus is slightly low in position. The arrow indicates a small area of calcification which was later expectorated. 3. The medial portion of the diaphragm is poorly outlined. 4. The mediastinum is displaced to the right.
Lateral roentgenogram: 1. The shadow of increased density is seen with its base obscuring the posterior half of the diaphragm. 2. The inferior half of the major septum shows a marked curve and is displaced posteriorly. 3. The area of calcification is present at the apex of the collapsed area. (Incidental collapse of the lingula of the left upper lobe had been present for years.)
Follow-up roentgenograms after expectoration of the calcified node showed marked improvement and nearly complete re-expansion of the right lower lobe.

against the major septum. The position of the superior portion of the major septum is greatly changed. It is much lower than normal, the angulation usually occurring in the mid chest—on the right side at the junction of the major and minor septa, on the left at approximately the same level. Ordinarily, the hilus is not appreciably depressed, but a small amount of compensatory emphysema in the adjacent portions of the upper lobe or in the basilar portion of the lower lobe may be demonstrated. It is of some importance to note that bronchiectasis may spare the dorsal division when the remainder of the lower lobe is involved, whereas tuberculosis and lung abscess frequently involve the apical portion of the lower lobe.

In the group studied, collapse of the basilar portion of the lower lobe was more frequently seen than collapse of the entire lobe or of its dorsal division. With future advance in thoracic surgery, further subdivision of the basilar portion will probably be required. Various classifications for such subdivision have been suggested (1, 2, 5, 6). The importance of classification is that there should be agreement between the roentgenologist and the surgeon. Due to lack of sufficient proof, no detailed discussion of the appearance of collapse of segments of the basilar portion of the lower lobe can be given (Figs. 4 and 5). There have been numerous instances in the presence of foreign body, so-called atypical pneumonia, and other disease processes, in which it was thought that there was atelectasis or collapse of one or more of these segments, but in very few was absolute confirmation obtained.

Actually the basal portion represents about two-thirds of the bulk of the lower lobe, and its appearance when collapsed (Fig. 6) is very similar to that of collapse of the entire lobe. It differs in that the shadow of increased density is somewhat smaller, and posteriorly, between it and the posterior chest wall, there is downward

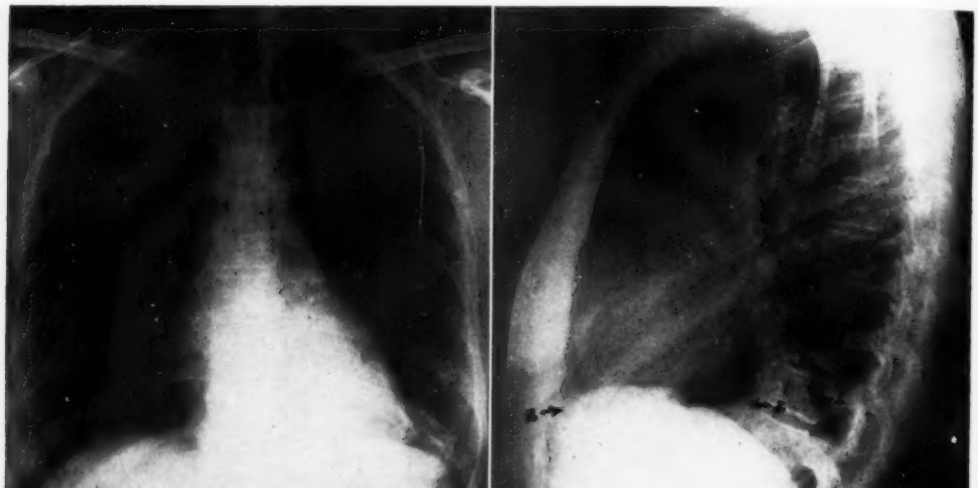

Fig. 6. Collapse of the basal segments of the left lower lobe due to mucous plug in the left lower lobe bronchus distal to the dorsal division bronchus.

Postero-anterior roentgenogram: 1. The shadow of increased density is visible through the heart shadow. 2. The hilus is only slightly low in position. 3. The left diaphragm is slightly elevated.

Lateral roentgenogram: 1. The shadow of increased density represents complete collapse of all of the segments of the lower lobe except the dorsal division. 2. The anterior portion of the diaphragm is clearly visualized, and the emphysematous upper lobe extends back to the major septum, which forms the anterior margin of the shadow of increased density.

extension of the emphysematous dorsal division. The shadow cast by the collapsed basal segments obscures only the posterior portion of the diaphragm and is not in contact with as much of the posterior chest wall as the shadow of the collapsed entire lobe. The middle lobe moves somewhat posteriorly, but less so than in complete collapse of the lower lobe. The hilus is often displaced inferiorly, but the amount of displacement is dependent upon the degree of compensatory emphysema in the dorsal division. In other words, the greater the emphysema, the less the inferior displacement of the hilus. As in collapse of the entire lobe, there may be less inferior displacement of the hilus if a tumor composes part of the shadow of increased density. The major septum is usually visualized without difficulty, but that portion below the dorsal division always lies posterior to its normal position. Frequently some limitation of motion, as well as slight elevation, of the diaphragm is evident.

Acute collapse of the dorsal division, of the basilar division, or of its subdivisions, is similar to acute collapse of the entire lobe in that it may be reversible, the changes being easily observed on serial roentgenograms.

Occasionally the collapsed segment of a lower lobe may be surrounded by emphysematous blebs arising from the involved pulmonary segment. When this occurs, it tends to limit somewhat the spatial rearrangement of the remaining uninvolved portions of the lobe and thereby may detract from the significance of the signs which have been used to determine collapse. Correct diagnosis will then depend largely upon the actual appearance of the shadow of increased density.

It has not been our intention to deal with collapse of different portions of the lung as a disease entity, but rather as a manifestation of various disease processes. Early diagnosis of a particular process, such as bronchiectasis or neoplasm, may depend on the early recognition of this

particular manifestation or sign. If any part of a lung appears smaller than normal, or fails to assume its share in the phases of respiration, collapse of that part should be considered. Unless complete examination of the chest (7, 1) has been made, the roentgenologist is not justified in reporting as negative (and then only with certain understood reservations) the examination of any patient who complains of frequent upper respiratory infection, chronic bronchitis, or persistent pain. In interpreting the roentgenograms of such a patient, any change from normal in the shape or position of the septa should be noted. If associated change in the position of the hilus or rearrangement of the vascular shadows suggesting interference with aeration occurs, collapse of some degree should be strongly suspected, and more careful observation may reveal the presence of an ill defined shadow of increased density representing a collapsed portion of the lung.

CONCLUSIONS

Collapse of a lower lobe has been a common finding in the group studied, being present in approximately two-thirds of the cases.

Rearrangement of the vascular shadows, depression of the hilus, and demonstration of an actual decrease in size of a lobe or segment, by means of changes in the position or contour of the septa, are of importance in the diagnosis of collapse of the lower lobes.

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The Value of Gastric Pneumography in Roentgen Diagnosis[1]

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THE ROENTGEN signs of disease in the pancreas, both benign and malignant, have been adequately covered by numerous writers. The complete review offered by Case in 1940 (3) leaves little to be desired, as he reviewed thoroughly all of the literature. More recently Borak (2) has also discussed the roentgen findings. The most striking fact apparent from these papers is the difficulty of making an early diagnosis of disease or tumor in the region of the body and tail of the pancreas. The diagnosis of enlargement of the head of the pancreas was well covered by Case (3) and subsequently by Rendich *et al.* (9). The routine methods of both clinical and roentgen examination may reveal the presence of a mass in the upper abdomen, but too frequently accurate localization of this mass is impossible, and the need for further information is felt. Gastric pneumography may be useful in just such cases.

Since 1933, when Engel and Lysholm (4, 5) introduced gastric pneumography as an aid in the diagnosis of pancreatic disease, the method has been widely quoted, but usually with an air of "this is included for the sake of completeness" (7, 9). Berk (1) in his review does not mention the procedure. No attempt has been made in the American literature to evaluate the method *per se*. Borak (2), in his review, agrees with Paul (8), whom he quotes: "The difficulty is that it is not applicable as a routine method. . . unless some positive findings are present, the examiner does not know which cases may need such an added examination." Paul himself introduces his comment by observing that "the early symptoms and signs [of carcinoma of the pancreas] are by themselves not distinctive and are similar to those associated with other intra-abdominal lesions. In certain cases the difficulties in arriving at a diagnosis may appear insurmountable." Where there is so much difficulty in arriving at a diagnosis, any additional information which can be obtained by a different method of examination must be worth seeking. This was pointed out by Case (3), when he wrote: "The method constitutes a very definite advancement in the roentgenography of the pancreas."

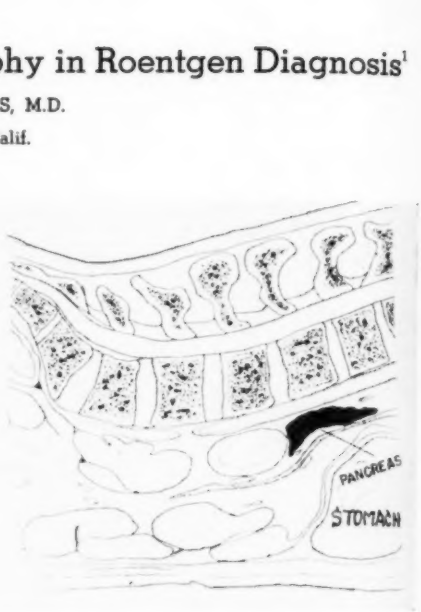

Fig. 1. Sketch (after Engel and Lysholm) of a sagittal section through the mid-line at the level of the pancreas and stomach, showing clearly the relationship of the two organs in the same projection as used in gastric pneumography.

With these thoughts in mind, we attempted to obtain gastric pneumograms in a series of 38 cases where the routine clinical and roentgen examinations pointed to a lesion of or near the body or tail of the pancreas, and some further aid in localization or diagnosis was needed. Review of these cases appeared to be worth while in order to emphasize the diagnostic points and bring this method of examination to the attention of those radiologists who have not already used it.

[1] From the Department of Radiology of Stanford University School of Medicine and San Francisco Hospital. Accepted for publication in October 1944.

TECHNIC

The use of any new technic is beset with difficulties, and all too often the procedure is discarded without full trial because of primary failures. The technic used by us differs from that of the original procedure described by Engel and Lysholm in 1933 and 1934 (4, 5). Their method of distending the stomach with effervescing powders and carbonated water was attempted, but the type of patient with whom we were dealing and the undesirable presence of the fluid in the stomach (Fig. 4) made us seek a variation. We made use of a small-caliber stomach tube, which served first for aspiration of gastric contents and then inflation of the stomach. The patient was laid prone with high supports under the chest and pelvis. This was done to insure a normal contour of the posterior wall of the stomach which silhouettes the retrogastric space. The beam of rays is horizontal, and a vertical Bucky diaphragm is used. The patient is centered before undertaking the last step, the insufflation of air into the stomach, which must be followed immediately by exposure of the film. The amount of air is not predetermined but is governed by the ability of the patient to restrain regurgitation. The quantity varies from 300 to 800 c.c. The smaller amount is not so satisfactory but may be used in less tolerant patients. The purpose of the immediate exposure of the films is to avoid filling the upper small bowel with air and thereby obscuring the outlines of the stomach. Only one or two exposures are attempted at a time. If these are unsatisfactory, one should not persist but should re-examine the patient at a later date. The exposure factors are those used for lateral gastro-intestinal films.

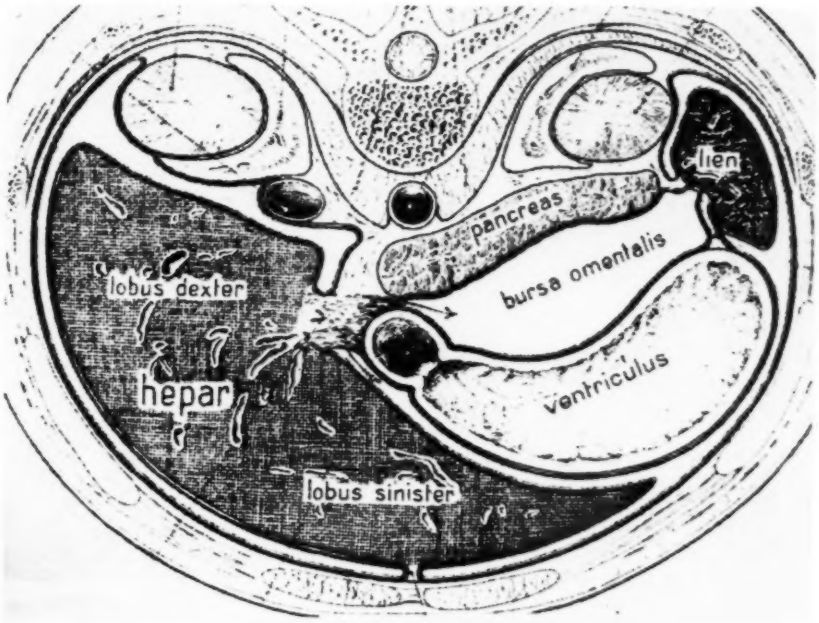

Fig. 2. Sketch (after Sobotta and McMurrich) of a cross-section at the level of the stomach and pancreas, illustrating further the relation of the pancreas to the stomach, as well as the relationship of the other upper abdominal viscera.

We do not feel that a postero-anterior view of the gas in the stomach is necessary. In all of our cases routine gastro-intestinal examinations had been done, and

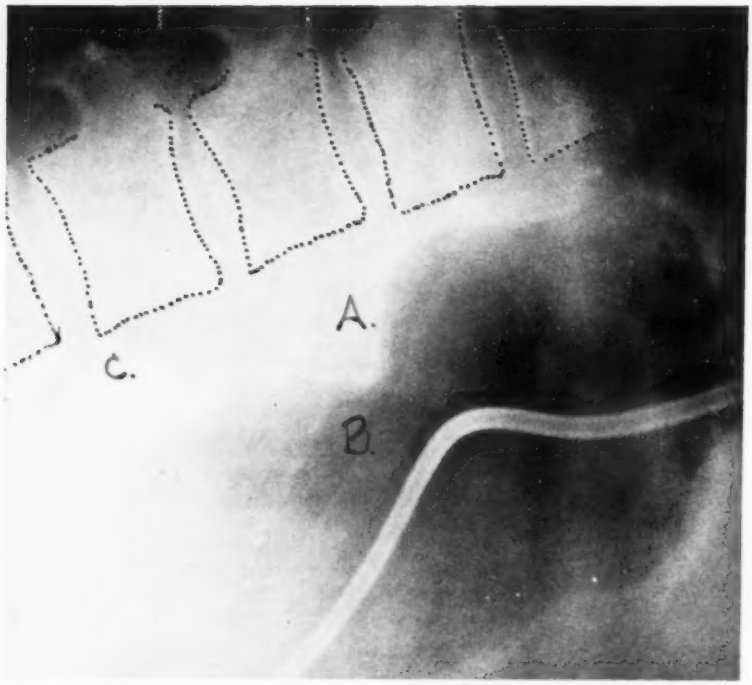

Fig. 3. Normal gastric pneumogram. G. W., 64-year-old male, complained of abdominal pain and weight loss of 20 pounds over a period of three months. An upper abdominal mass 12 cm. in diameter was felt. Gastro-intestinal studies showed no displacement of the stomach and it was felt that the mass was in the left lobe of the liver, lying slightly anterior and to the right of the stomach. Pneumograms of the stomach revealed no enlargement of the retrogastric space. The outline of the pancreas appeared normal. The patient died two weeks after the examination. A hepatoma was found in the left lobe of the liver. The pancreas was normal in size, shape, and position.

A. Silhouette of retrogastric space (a normal pancreatic shadow).
B. Stomach distended with air.
C. Gas in duodenum and barium in colon.

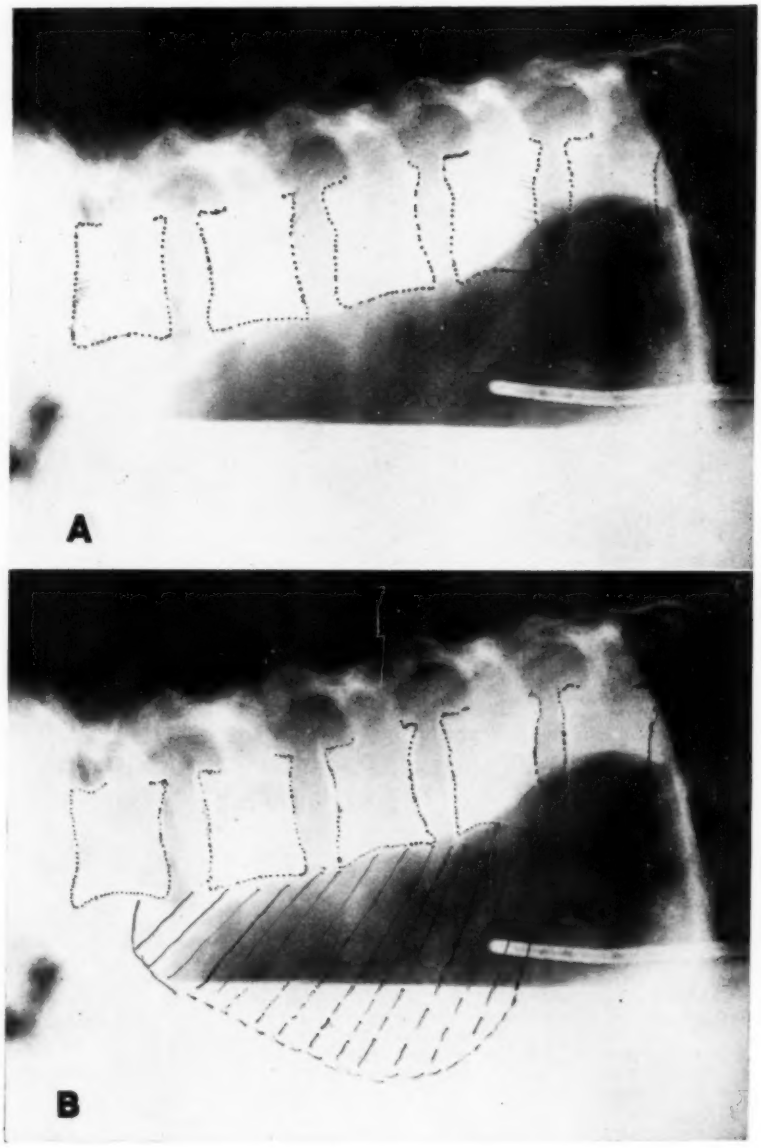

Fig. 4. A. Pneumogram with fluid in the stomach. H. N., 63-year-old male, complained of progressive weakness for four months and jaundice for ten days. Physical examination revealed a large nodular liver and epigastric mass. A gastric pneumogram was reported as "not revealing any increase in the retrogastric space." Autopsy, a few days later, revealed a large carcinoma of the body of the pancreas, which had spread to the draining lymph nodes and to all the abdominal viscera. The tumor with the involved nodes created a large retrogastric mass. This was not outlined pneumographically because the fluid in the stomach dammed off its distal portion.
B. Reconstruction of the shadow which was not shown.

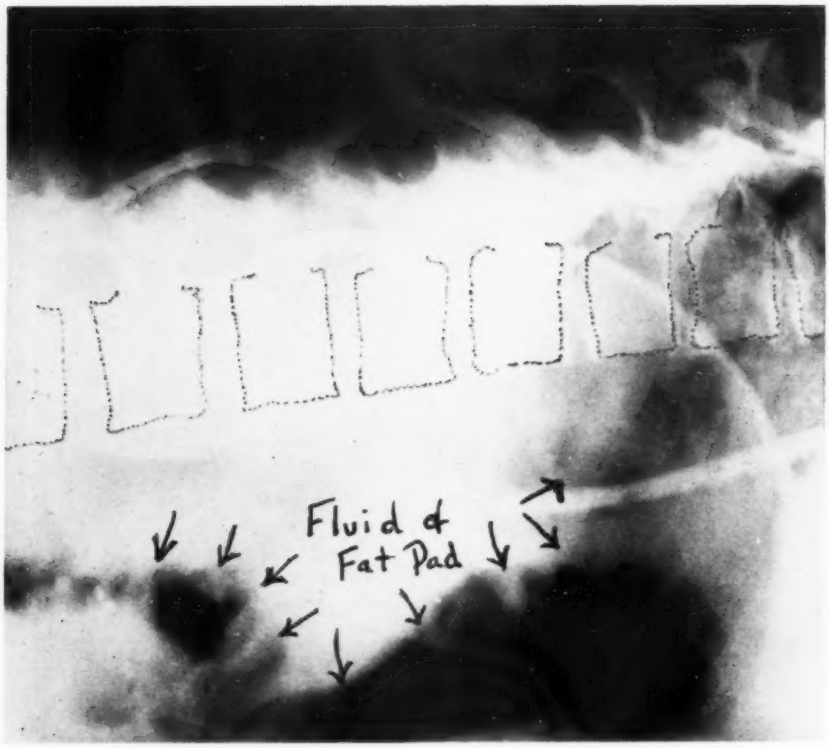

Fig. 5. Fluid and fat in the lesser omental sac. M. A., 48-year-old female, had jaundice of five weeks' duration. The liver was barely palpable and smooth. No epigastric masses were felt. No signs of peritoneal fluid were elicited. Gastro-intestinal examination showed the stomach to be displaced anteriorly and upward. The duodenal loop was exaggerated. The second portion of the duodenum never completely filled and there was a 10 per cent gastric residue at six hours. Gastric pneumograms revealed increase in the retrogastric space which was taken to indicate an enlarged pancreas. A diagnosis of tumor in the pancreas was made.

At operation, cirrhosis of the liver was found with gross enlargement of the liver, fluid, and a large fat pad in the lesser omental sac, the combination producing the expansion in the retrogastric space demonstrated on the film. The pancreas was normal in size, shape, and position.

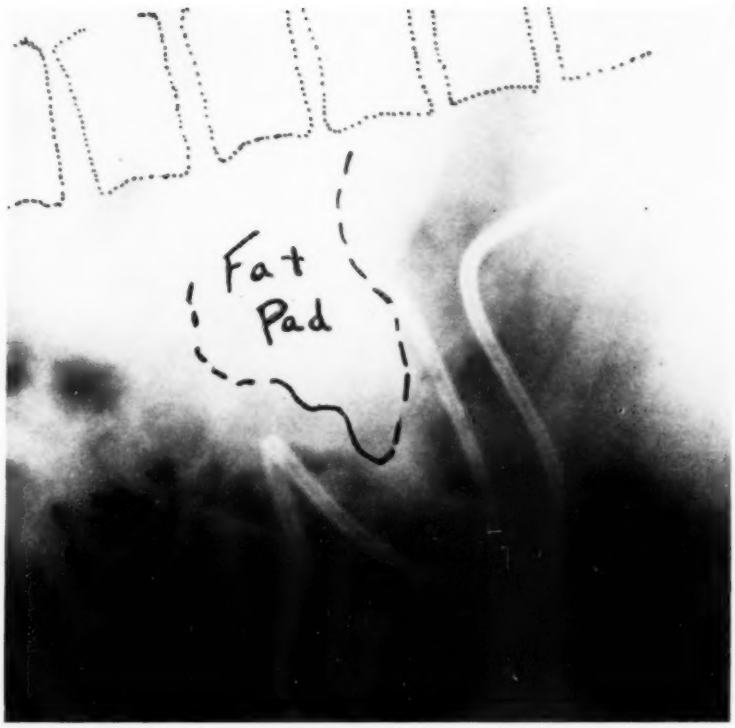

Fig. 6. Fat pad in lesser omental sac in a very short obese patient. G. M., 31-year-old male, entered the hospital with the diagnosis of an "epigastric mass, probably pancreatic cyst." He had had upper abdominal pain intermittently over a period of two years. Gastro-intestinal studies were felt to confirm the impression of a pancreatic mass. Gastric pneumograms showed a marked increase in the thickness of the retrogastric tissues, which was felt to be still further confirmation of a pancreatic mass.

At operation a large lesser omental sac fat pad was found to be the cause of the increase in the thickness of the retrogastric tissues. No cause for the upper abdominal pain was found.

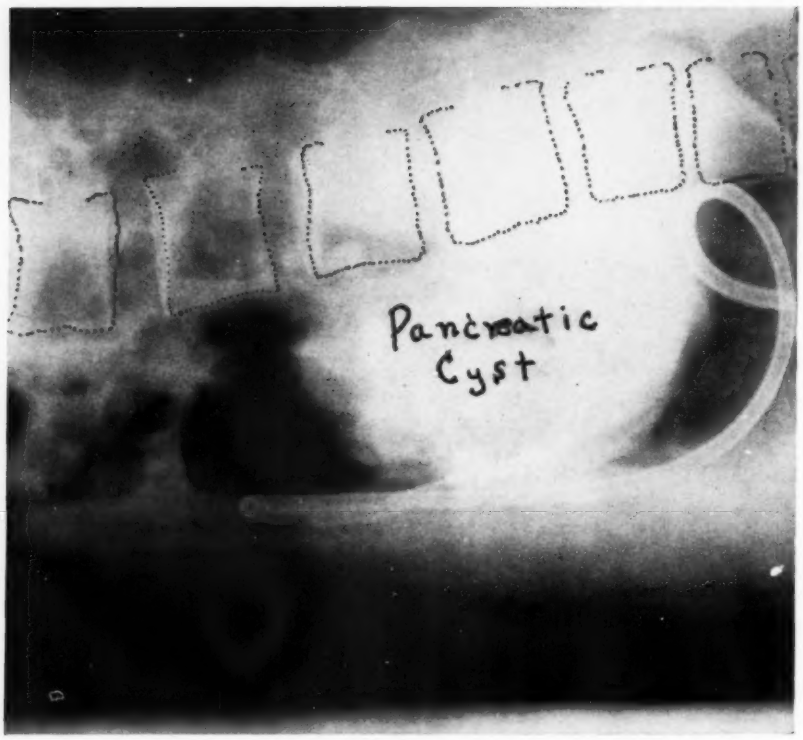

Fig. 7. Enlargement of pancreas, pancreatic cyst. G. G., 31-year-old male, entered the hospital after an alcoholic spree. He had had five previous hospital entries with alcoholism and occasional bouts of epigastric pain. A gastro-intestinal examination had been done on the next to the last admission, and a suggestion of pancreatic enlargement made. The symptoms and sign (serum amylase) returned to normal, however, and the patient was discharged.

On entry two months later, a gastric pneumogram showed a great increase in the retrogastric space, and the serum amylase went to 364 units. On the night before operation, the patient had a severe abdominal pain and almost went into shock. At operation a ruptured cyst was found in an indurated enlarged pancreas which contained many parenchymal calcifications. The calcifications cannot be seen on the reproduction, but the obviously enlarged retrogastric space caused by the pancreatic cyst is clearly demonstrated.

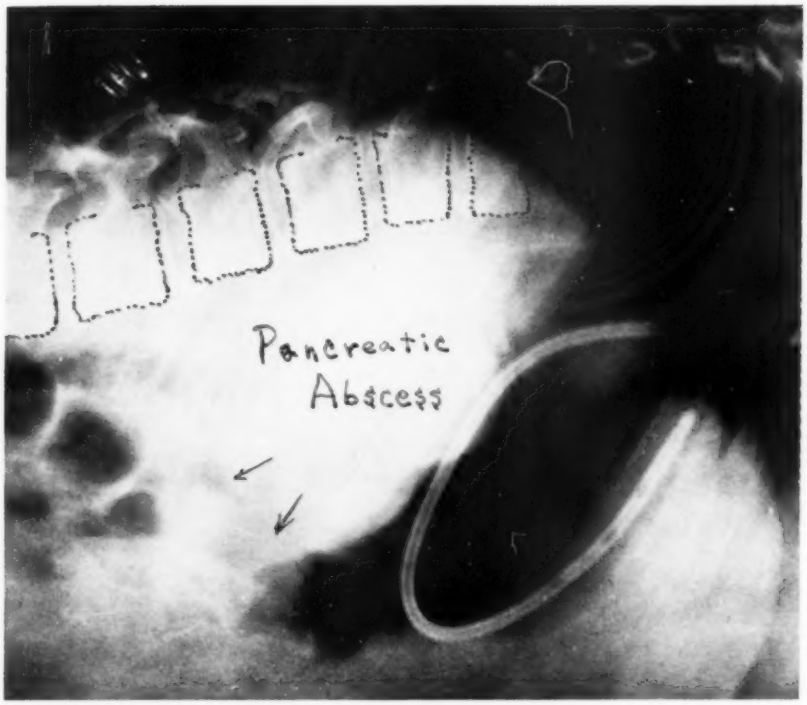

Fig. 8. Enlargement of pancreas, pancreatic abscess. H. S., 29-year-old female, complained of abdominal pain for one day following an alcoholic spree. On physical examination her abdomen was tense, with tenderness and guarding in the upper quadrants. The white blood count was only 9,000. The pain subsided in twenty-four hours, but the patient went into an acute bout of delirium tremens, despite sedation and fluids. She was sent to the psychiatric ward for further observation and treatment. As she cleared mentally, the abdominal pain returned and the serum amylase was found to be 313 units. Finally, two weeks after entry, an upper abdominal mass was palpated. Gastric pneumograms showed a tense, lobulated mass posterior to the stomach. At operation a pancreatic abscess was drained. The fluid from the abscess was tested for amylase and found to give a reaction of 5,000 units. A culture showed *E. coli*. After a stormy postoperative course, with formation of a right lower quadrant abscess which necessitated drainage, the patient recovered fully.

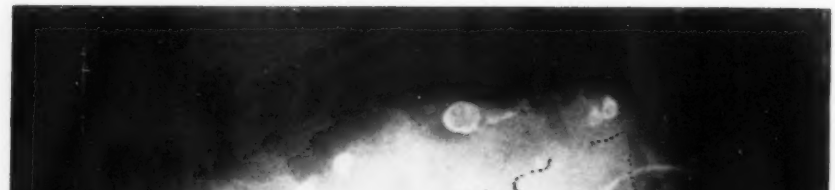

Fig. 9. Enlargement of pancreas, carcinoma in tail of pancreas. C. S., 64-year-old male, gave a history of pain in the left upper quadrant and epigastrium for one month. He had lost 24 pounds. On physical examination a 7 × 4.5-cm. mass could be felt. It was firm, slightly irregular, and tender. The liver was also irregular and tender. The clinical impression was a retroperitoneal tumor, but following a gastro-intestinal series, a diagnosis of pancreatic or gastric tumor was suggested. The gastric pneumogram shows the large mass in the region of the pancreas. The barium-filled diverticula in the colon are also seen.

At laparotomy a diagnosis of carcinomatosis was made, probably arising from the pancreas. The patient died three days after operation and at autopsy a large carcinoma was found originating in the tail of the pancreas with spread to practically all the abdominal viscera.

the position and shape of the stomach were known.

INTERPRETATION

The anatomy of the region about which we are concerned is best shown by topographical sketches in two planes (Figs. 1 and 2), as found in Engel and Lysholm's original article and Sobotta and McMurrich's *Atlas* (the latter reproduced by Borak).

Engel and Lysholm believed the normal thickness of retrogastric tissues was the depth of the vertebral body lying posterior to the stomach; in other words, the depth of the first or second lumbar vertebral body as seen in the lateral projection (Fig. 3).

Fluid in the stomach sufficient to dam off its distal portion makes the interpretation of the film more difficult and may easily lead to errors (Fig. 4). It is only with practice and evaluation of the patient as a whole (especially the body build) that approximate normal standards may be evolved. Holm (6) reviewed some 449 cases and found that (1) obesity (Fig. 6) and (2) ascites (Fig. 5) produced an increase in the thickness of the retrogastric tissues which simulated pancreatic enlargement. He also lists other conditions which may produce anterior displacement of the stomach: (1) tumors of the liver, (2) metastatic nodes, (3) infiltration of the pancreas due to perforated ulcer, (4) hypernephroma and retroperitoneal neoplasms. Holm's work does not point out that, although these conditions do produce an increase in the retrogastric space, actual pancreatic disease produces a clear-cut appearance (Figs. 7, 8, and 9)—so clear-cut that we have had no difficulty in those cases in which the disease lay wholly in the pancreas. The enlargement may be due to inflammatory, cystic, or neoplastic change. Our errors in interpretation have been in those cases in which fluid or a fat pad widened the retrogastric space and simulated pancreatic enlargement (Figs. 5 and 6).

Holm also reported the diagnosis of atrophy of the pancreas by a marked decrease in the retrogastric tissues, but we have not seen any such cases.

SUMMARY AND CONCLUSIONS

A simplified technic for gastric pneumography is presented, and the interpretation of normal and abnormal findings is discussed.

Gastric pneumograms are of definite supplementary diagnostic value in the location and diagnosis of enlargement in the body and tail of the pancreas (both benign and malignant) and of aid in the localization of certain epigastric masses.

NOTE: On page 204 of this issue of RADIOLOGY is an abstract of a paper, from the surgeon's point of view, including ten case reports which were reviewed at the time this communication on the roentgen aspects was written.

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Rapid Development of Bone Changes in a Patient with Syringomyelia as Observed Roentgenologically[1]

EUGENE P. PENDERGRASS, M.D., GEORGE D. GAMMON, M.D., and LT. JAMES H. POWELL, M.C., A.U.S.

WE HAVE HAD under observation a patient with syringomyelia, who within a few weeks developed marked bone changes in several joints. This case is being reported to illustrate the rapidity with which these changes may occur and to emphasize, among other things, their importance in neuropathic joints because of the medicolegal and compensation aspects.

CASE REPORT

O. H., a 44-year-old Negro, was referred for a roentgen examination of a painful left shoulder on Feb. 17, 1942. A diagnosis of subdeltoid bursitis had been made in the surgical outpatient department and novocain injections had been given to relieve the pain. The roentgen examination showed nothing abnormal (Fig. 1).

A month later (March 16, 1942) the patient returned to the surgical outpatient department because of an elevation of temperature, ranging to 102°. The shoulder ached and was swollen and fluctuant. It could be dislocated downward and posteriorly and the dislocation could be reduced without pain. Aspiration yielded about 50 c.c. of serosanguineous fluid. Smears and cultures were negative. A roentgen examination, March 24, showed considerable destruction of the head of the humerus, and a diagnosis of a neuroarthropathy was made (Figs. 2 and 3). Up until this time the previous history of the patient was entirely unknown to members of the department of roentgenology.

A request was made for an examination of other parts of the body. The only other abnormal joints observed were the right and left elbows, which showed beginning absorption of cartilage and some hypertrophic changes (Figs. 4 and 5).

The patient was an alum packer and had been admitted to the neurology clinic in 1927, complaining of soreness and numbness in his left hand, especially along the distribution of the ulnar nerve. In the next three years his symptoms progressed. The left pupil was larger than the right. Serologic tests of the blood and spinal fluid were negative. By 1940, a limp and weakness of the left knee had developed. Amputation of the right middle finger was done for a non-healing ulcer. Serologic tests were again negative.

Fig. 1. Roentgenogram of left shoulder girdle, Feb. 17, 1942, showing nothing abnormal; no roentgen evidence of bursitis.

In December 1941, the reflexes were recorded as follows:

Biceps
Right: Markedly depressed
Left: Markedly depressed

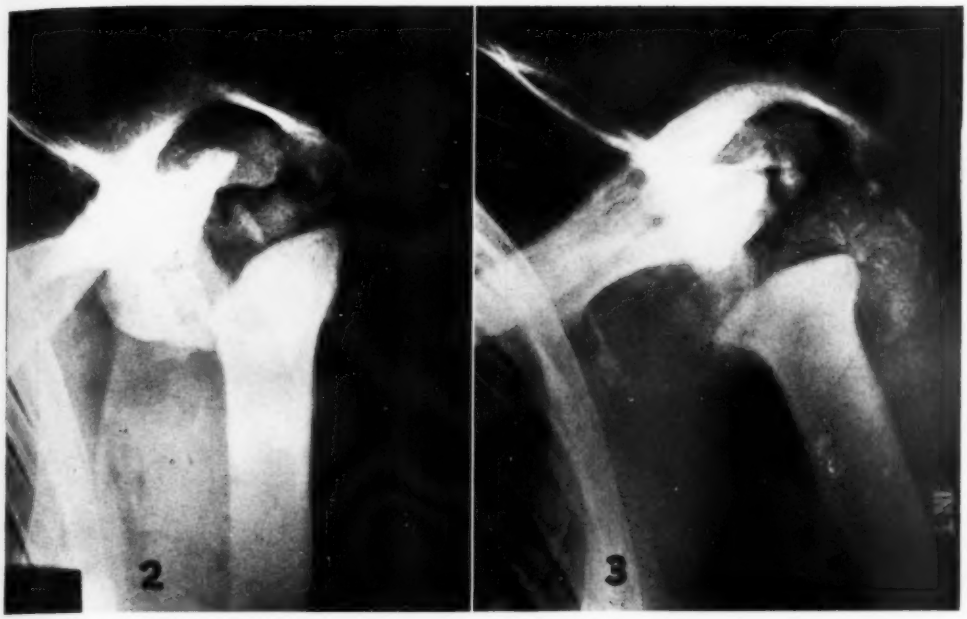
Triceps
Right: Markedly depressed
Left: Markedly depressed

Abdominals
Right: Absent
Left: Absent

Cremasteric
Right: Exaggerated
Left: Exaggerated

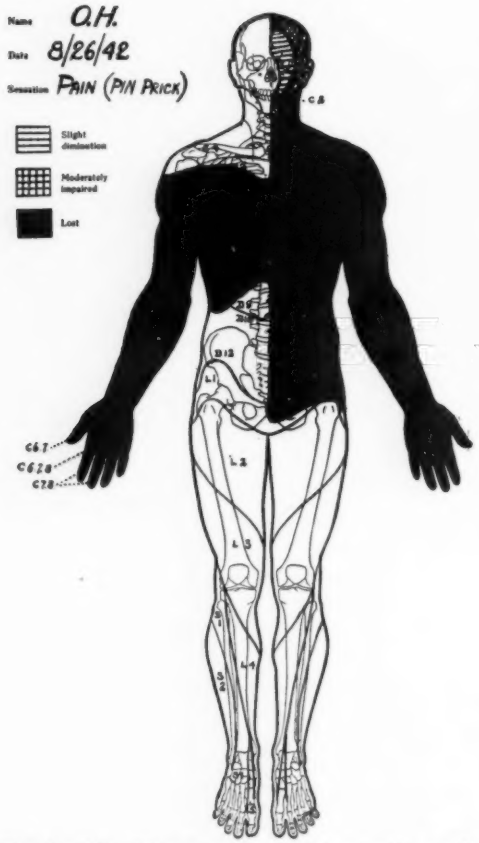
Patella
Right: Exaggerated
Left: Greatly exaggerated

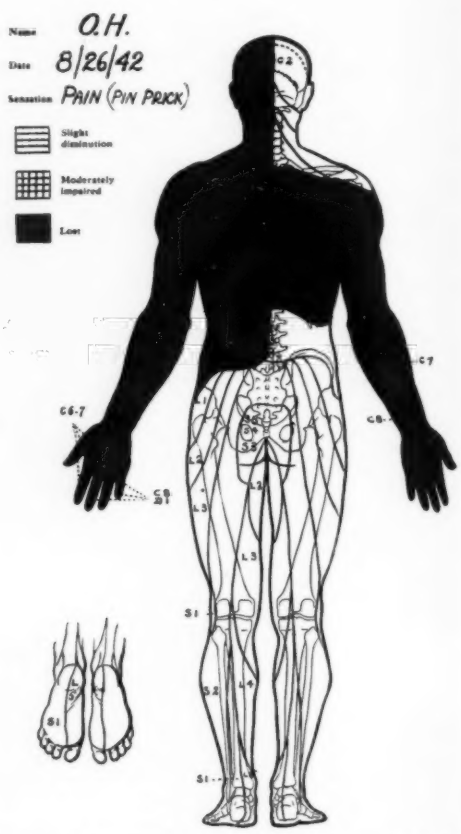
[1] From the Departments of Radiology and Neurology in the Hospital of the University of Pennsylvania, Philadelphia, Penna. Accepted for publication in December 1944.



Figs. 2 and 3. Roentgenograms in internal (Fig. 2) and external (Fig. 3) rotation, made March 24, 1942, approximately five weeks after the original examination (Fig. 1). The head of the humerus is completely destroyed and there is considerable bony debris in the joint. The glenoid fossa of the scapula is also involved. Many studies have been made of this shoulder over a two-year period and the bone destruction has increased only slightly.

Figs. 4 and 5. The left elbow (Fig. 4) shows evidence of cartilage destruction, especially on the external condyle of the humerus. There are loose fragments of bone in the soft tissues and a peculiar overgrowth of the lateral aspect of the head of the radius. Changes in the right elbow (Fig. 5) are less marked than in the left. In the lateral view bone fragments are seen in the soft tissues.





Figs. 6 and 7. Diagrams of loss of pain sensation on the anterior and posterior aspects of the body, Aug. 26, 1942.

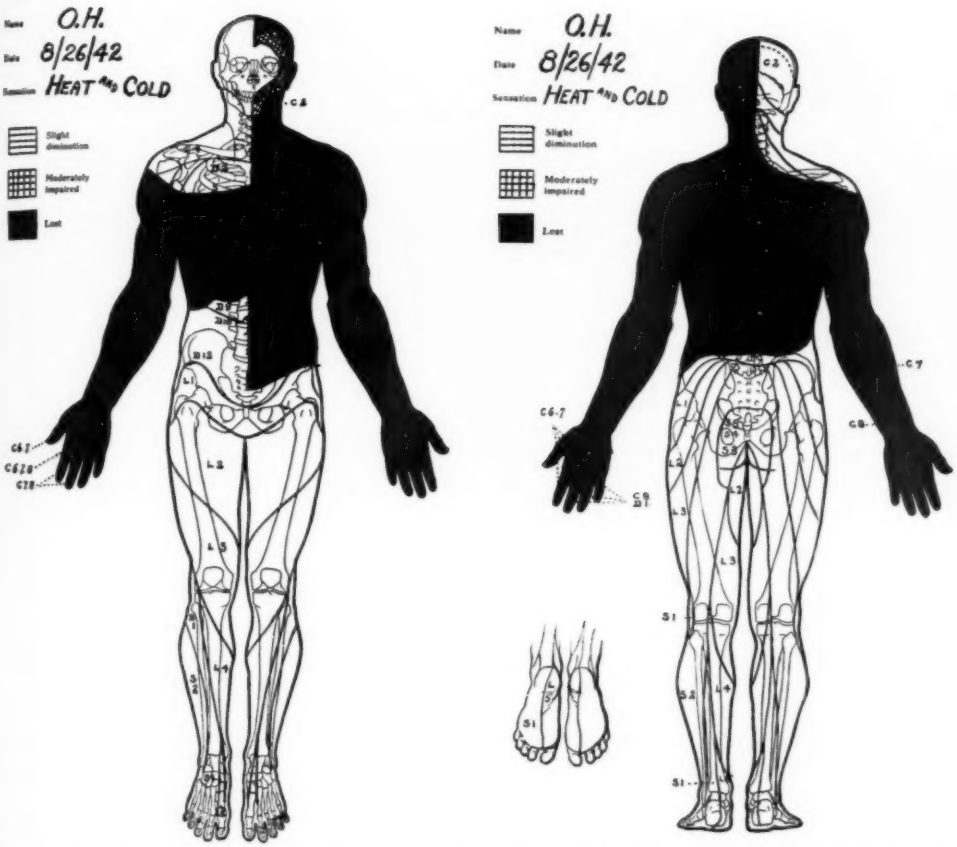
Achilles
Right: Exaggerated
Left: Greatly exaggerated
Babinski
Right: Negative
Left: Negative
Patella clonus
Right: Present
Left: Sustained
Ankle clonus
Right: Sustained
Left: Sustained

Serologic tests of the blood and spinal fluid were still negative. The colloidal gold curve was flat.

After the roentgen diagnosis, another effort was made to determine the cause of the patient's illness. A provocative test for syphilis was started on June 30, 1942, and continued for one month. Repeated serologic tests were negative.

In August 1942, the physical examination revealed scoliosis of the lumbar spine with the convexity to the left; shortening of the left arm, with decrease in tone of all of the muscles of the arm; slight wasting of the rhomboideus, supraspinatus, and infraspinatus muscles of the left shoulder girdle, with no other muscular atrophy. Muscular weakness was observed in all extremities, more noticeable on the left, especially in the left hand. The gait was spastic, the left leg exhibiting the greater spasticity. Romberg test negative. Reflexes as follows:

Biceps
Right: Absent
Left: Absent
Triceps
Right: Barely perceptible
Left: Absent
Supinators
Right: Absent
Left: Absent
Abdominal (upper and lower)
Right: Weak
Left: Absent



Figs. 8 and 9. Diagrams of loss of heat and cold sensation on the ventral and dorsal aspects of the body, Aug. 26, 1942.

Cremasteric
Right: Normal
Left: Absent
Knee jerk
Right: Exaggerated
Left: Spontaneous clonus
Ankle jerk
Right: Exaggerated
Left: Greatly exaggerated
Patella clonus
Right: Sustained
Left: Markedly sustained
Ankle clonus
Right: Sustained
Left: Markedly sustained
Babinski
Right: Negative
Left: Positive

There were diminution of pain sensation over the left trigeminal area, loss of pain sensation with preservation of touch from segment C_2 to T_{12} on the left, and similar findings on the right from segment C_5 to T_{10}. Sensation of heat and cold was lost over essentially the same areas as pain (Figs. 6–9). Vibratory sense, sense of position, graphesthesia, light and heavy touch sensation were all normal.

Cranial nerve examination showed no involvement of the olfactory sense. Visual fields and retinae were normal. There was no ptosis of the lids. The left pupil was larger than the right and reacted sluggishly to light. There was rotatory nystagmus on left lateral gaze. The left corneal reflex was barely perceptible. Sweating was much more profuse on the right side of the face. There was sensory diminution, as noted above, over the left trigeminal area. The patient was slightly hoarse, but direct laryngoscopy failed to reveal any abnormal cord changes or evidence of paralysis.

Spinal tap yielded a clear fluid under a pressure of 170 mm. water, with 4 white blood cells per cubic millimeter, 25 mg. per cent protein, a negative Wassermann reaction, and a colloidal gold curve of

Fig. 10. Roentgenogram of left shoulder, July 19, 1943, seventeen months after original examination (Fig. 1). Bone destruction in the scapula and humerus has increased only slightly.

0000000000. The Queckenstedt test was normal. The blood sedimentation rate was 9 mm. per hour (normal according to our standards), serum calcium was 9.5 mg. per cent, serum phosphorus 3.7 mg. per cent, and serum phosphatase 7.2 alkaline and 0.5 acid (Shinowara, Jones and Rhinehart units). A routine blood count, hemoglobin determination, and urinalysis all showed findings within normal limits.

DISCUSSION

It is our opinion that this patient has syringomyelia with multiple neuropathic arthropathies. Our interest is largely in the following questions:

1. What is known about the rapidity of changes in a neuropathic joint?
2. Why do pathologic changes progress to a certain point and then become relatively stationary (Fig. 10)?
3. What is the medicolegal status of such a case?

In the beginning, neuropathic joints were thought to occur only in tabes dorsalis, and the condition of neuroarthropathy, even today, is commonly referred to as "Charcot's joints." It is now known that neuropathic joints occur in other conditions that lead to destruction of cord areas and possibly in association with peripheral nerve lesions. Thus the process may perhaps better be referred to as a "neuroarthropathy" until the correct diagnosis is established.

Charcot (2) described the development of neuropathic joints in 1868 and stated that the changes were due to neurotrophic and circulatory disturbances from cord degeneration. Other writers, as Eloesser (6), Corbin and Hinsey (4), Wile and Butler (21), believe in an added traumatic factor.

Soto-Hall and Haldeman (18) have outlined in simple form the various steps in the development of a neuropathic joint, with the essential etiological factor as the only definite variable in the schema. Eloesser's studies (6) of normal cats under controlled experimental conditions show that an anesthetic joint alone or a damaged joint later denervated will not go on to produce an arthropathy, but anesthetic joints subjected to trauma will, in 100 per cent of cases, become characteristic Charcot joints within a period of three weeks.

Approximately 10 per cent of patients with tabes are said to have arthropathies, and the joints of the lower extremities are involved in 75 per cent of this group. In syringomyelia about 25 per cent of the patients have neuropathic joints and approximately 80 per cent of these show involvement of the upper extremities (20). This distribution of the lesions is explained on the basis of the early involvement, in tabes, of the lumbar and lower thoracic portions of the spinal cord, whereas in syringomyelia the process is more likely to occur in the lower cervical and upper dorsal regions.

Neuropathic joints are generally, though not invariably, painless. In our case the pain was such that a presumptive diagnosis of subdeltoid bursitis was made. The persistence of pain is due to over-distension of the more superficial tissues, the sensation in the bone, joint, and periosteum having been lost. Eloesser (7) assumes that there are different spinal tracts for the pain fibers from bone and periosteum than from the skin.

Rapidity of Development of Joint Changes: It is a common supposition that the osteoarthropathies of syringomyelia develop

more slowly than those of tabes. In our patient the clinical evidence of joint destruction became obvious in less than three weeks and was recorded roentgenographically within five weeks (Figs. 2 and 3).

Charcot (2) believed that the onset was always sudden. He presented the Museum of the Royal College of Surgeons (16) with a specimen, a humerus, showing atrophy of the head though the neck was intact. This syphilitic arthropathy was reported to have developed over a period of three months and was used by Charcot in his lectures to illustrate the rapidity of the process. Others have not substantiated this view of rapid development. Very few have described any effort to determine the time between the so-called normal and the full-blown neuropathic joint.

Wile and Butler (21) presented 88 cases of Charcot's arthropathies, of which only 9 showed a "rapid" onset. They were convinced that the mode of onset is a gradual one, that months or even years may elapse before the full-blown Charcot joint develops, and that the occasional apparently sudden production of an arthropathy is frequently the result of trauma, bringing under consideration a pre-existing subclinical process.

Brailsford (1) followed roentgenologically the development of a neuropathic shoulder joint which required twenty months. Neurologic signs did not develop until three years later. Key (12) followed a normal tabetic hip through the Charcot joint stage in nine months. Philips and Rosenheck (14) describe an arthropathy observed roentgenographically six months after a wrenching accident to the part. Funsten (8) reports the "rapid development" of a tabetic arthropathy of the spine over a nine-month period, and Chitty (3) reports a case with radiological records of the development of a Charcot hip within five months after a pelvic fracture. In Horwitz's patient (11), a fifty-year-old woman who had injured her ankle, but with no fracture or roentgen evidence of bone changes, a Charcot joint developed in four months.

Cotton (5) reports a tabetic arthropathy in a fifty-six-year-old woman eight weeks after a fracture of the styloid process of the radius, but the roentgenogram at the time of the original injury showed some "obvious pathological changes" in addition to the fracture. Potts (15) presents the case of a seventy-one-year-old male who already had Charcot joints, who suffered a traumatic fracture of the shaft of the humerus. Five weeks after the fracture, an enormous amount of callus was present in and around the painless break, leading to a pseudo-joint of the Charcot type. Halstead and Dick (9) report the development of a Charcot joint within ten weeks after trauma to an elbow in a patient with syringomyelia.

Eloesser (6), in an effort to evaluate the time element in the development of neuroarthropathies, subjected a number of normal, healthy cats to root section, preserving motor function but eliminating limb sensation. An operative deforming arthritis was then produced in the anesthetic joint by cautery. The cats were allowed to continue their normal activities as best they could, and in every animal a typical Charcot joint with effusion was present within three weeks.

One wonders, therefore, whether the relative rapidity of development of a neuropathic joint is dependent on the underlying disease condition, or whether this concept should be revised and all neuroarthropathies, regardless of etiology, be regarded as an entity, with the speed of development dependent upon the completeness of the anesthesia of the joint, the degree of activity of the patient, and the severity of the trauma.

In our patient, we were unable to determine any unusual trauma, but there had been injection of novocain into the joint. The question arises not only as to whether this procedure can, under such circumstances, be regarded as traumatic but also whether the anesthetic effect of the novocain was an activating factor or merely coincidental.

Up until the time our patient came under observation, it was our opinion and that of

many of our medical and surgical colleagues that the development of neuropathic joints was gradual. None of us suspected that there could occur such rapid destructive changes. One supposes that the rounded smooth ends of the affected bone may have been responsible for the concept of a prolonged process. Since seeing our patient, we have heard of two cases of neuropathic ankle joints which developed within a few weeks following fractures in that region.

Extent of Involvement: In considering why the disease process, in most instances, proceeds to a certain point and then stops, Potts (15) and Steindler (19) state that the bone destruction ceases when the bones fail to articulate or when invalidism occurs. In a discussion (19) of the development of neuropathic joints, it is stated that pathological fractures may be the first sign of an oncoming arthropathy. These fractures occur in or near a joint; they are characteristically transverse (as opposed to the oblique and spiral fractures seen in osteoporosis) and tend to heal rapidly. The changes begin in the cartilage and in the calcification zone between the cartilage and the underlying cancellous bone. Microscopically, in the beginning, there is cartilaginous hyperplasia with increased hyaline substance, followed by the appearance of cavities and fissure-like spaces in which connective tissue and fibrillae are matted together in the intermediary calcification zone. A pannus then covers the cartilage, which is resorbed from above. At the same time, cartilage catabolism occurs from below, through the marrow spaces of the spongiosa. In this way, hollow spaces, the so-called Weichselbaum lacunae, are formed and the cartilage appears honeycombed. The zone of preliminary calcification is then rejuvenated, and vascular marrow spaces push up into the cartilage. Thus an increasing disturbance of the joint structure occurs, with simultaneous proliferation and absorption. The cartilage becomes thinner, and the zone of increased calcification becomes thicker. The non-calcified cartilage wears away, the calcified cartilage is chipped off, and finally the irregular zone of calcification of the subchondral spongious substance lies bare and sclerotic bony facets are formed. By friction and concussion, a considerable number of these are destroyed and the contour and configuration of the joint are completely changed and irregular.

Trauma causes radial infractions through this very brittle preliminary calcification zone, leading to rapid callus and bone formation as repair from subchondral marrow zones occurs. These fractures lead to deformities, depressions, and displacements within the joint itself.

The synovia (13) becomes thickened and even calcified, and calcification may occur in muscles and tendons around the joint along the lines of greatest stress in trabecular structure. The points of stress show osteosclerosis, while points of relaxation exhibit osteoporosis.

When, therefore, an affected joint is splinted mechanically, or put at rest because of invalidism, and the osteosclerosis reaches a point which resists the ordinary stresses and strains, it is assumed that progression of the arthropathy ceases.

What happens to the muscles formerly attached to those areas of bone which disappear? This question cannot be answered fully, since no adequate necropsy studies have been published. In the case reported here, the entire shoulder girdle was swollen, some of the tumefaction being due to edema of the muscles and the surrounding connective tissues, and some due to abnormal shortening of the muscles, the result of decreased linear measurements between points of origin and insertion. This acute swelling may occur in a matter of hours, with or without trauma, decreasing only after weeks or months, when the chronic stage of disuse atrophy becomes manifest. The periosteal attachments are presumably intact, but the periosteum itself is ballooned out with synovial fluid, which is viscid and often bloody. The blood comes from the contused hypertrophied synovial villi, and King (13) states that the joint fluid is actually connective tissue with a fluid intercellular matrix.

Medicolegal Aspect: The medicolegal and compensation aspect of our case and of others with histories of recent trauma is one for serious consideration. It is stated that a Charcot arthropathy may be the result of a single severe trauma, especially one producing a fracture of the shaft of a bone or a fracture in or near a joint. Such cases have been reported as occurring in persons not known to have a pre-existing disorder such as tabes dorsalis or syringomyelia. Many writers have stressed the role of multiple minor forms of trauma as the important and most common factor in the production of Charcot joints, blaming even the shocks and strains of everyday life falling upon a joint unprotected because of its insensitivity.

When such a condition as Charcot joints develops as a result of an industrial accident, is the employer liable for the development of an arthropathy? According to Heyman (10), the rulings of the United States Supreme Court and the Federal Courts in cases of the "single, severe trauma" type give the injured man "a valid legal claim for the resulting disability when it would not have resulted had a previously existing disease not been present." Thus, where a disease is caused by an accident, the employer is liable, even though the accident would not have caused a similar disease in a normal person. Heyman further draws an analogy: "The fact that a man has abscessed teeth or diseased tonsils contributing to the absorption of 'toxins' does not exclude him from a just claim should he develop an osteoarthritis following an injury. Neither should compensation be denied the patient with an inactive syphilitic condition who develops a Charcot joint which appears immediately following an undoubtedly severe injury received in the pursuance of his occupation."

In a case reported by Sherwood and Hutchins (17) the patient suffered a fracture while at work and received compensation for a subsequent Charcot joint.

There have been no medicolegal complications in our case. There was no single severe trauma, but the patient's occupation requires handling of heavy packing cases. Our interest in the medicolegal aspects was stimulated by another case, clinically diagnosed as osteogenic sarcoma. The patient stated that his trouble was due to an injury sustained ten days prior to his admission to the hospital. Roentgenograms showed the typical changes of a Charcot joint (Fig. 11). Subsequently the clinical and serological findings showed that the patient had tabes dorsalis.

Fig. 11. Charcot joint in a patient with tabes dorsalis. According to the patient, the hip was all right until an injury ten days before.

Had we not had the opportunity of studying the case of syringomyelia, we would have pooh-poohed the idea that changes such as seen in Figure 11 could occur in such a short time. Now there is some question in our minds. The patient with tabes dorsalis was a truck driver and had no complaints until he was jammed against a building by his truck. Even then he did not concern himself until a swelling developed in the region of the hip.

COMMENT

It is hoped that this report may be of sufficient interest to stimulate radiologists,

whenever they encounter a Charcot joint, to obtain a careful history concerning trauma and time of development of the joint changes.

Our case of syringomyelia and some of those reported point to the necessity of obtaining past medical histories and neurologic examinations even in patients who have presumptive conditions such as subdeltoid bursitis, fracture, or bone tumors. Destructive changes may in many instances, in certain joints, develop more rapidly than is commonly suspected.

SUMMARY

A case of syringomyelia, with the development of extensive changes in the scapula and humerus of the left shoulder, is reported.

The changes appeared within three weeks after an injection of novocain into the tissues for the relief of pain thought on preliminary examination to be due to a subdeltoid bursitis.

Some of the literature reviewed indicates that severe trauma or minor injuries often repeated may result in the rapid development of neuroarthropathies.

Reports are available suggesting that injuries occurring prior to the development of neuropathic joints may have medicolegal and compensation importance.

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Unusual Pleural Effusions[1]

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THE TYPICAL roentgenogram in cases of pleural effusion is quite familiar. Briefly, the effusion in the absence of pneumothorax first produces a uniformly dense opacity obliterating the costophrenic angle. The medial border is crescentic and extends downward and inward toward the diaphragm. As the effusion increases, it extends medially over the diaphragm and upward along the lateral aspect of the chest. The free margin is concave and the density is higher in the lateral or costal portion of the thorax than in the medial. In the absence of a fixed mediastinum, large pleural effusions cause displacement of the mediastinal contents to the opposite side.

The upper concave margin seen in the roentgenogram does not, however, represent the actual height of the fluid. Kaunitz (1, 5) has demonstrated that simple pleural effusions may be divided into three regions. The lowest and most radiopaque zone is made up almost entirely of fluid, the middle or translucent area is due to a moderate amount of fluid, slightly compressing the lung, and the uppermost area is transparent but represents a film of fluid that is too thin to be seen in the x-ray film.

Small collections of fluid may not be seen in the conventional upright postero-anterior roentgenogram, the effusion being hidden in the posterior and anterior costophrenic sulci by the liver and the diaphragm. Thus Kaunitz (1) injected 300 c.c. of normal saline into the pleural cavity of an adult and was unable to obtain a shadow in the lateral costophrenic angle. Ganter (2) has shown that less than 400 c.c. of pleural fluid cannot be demonstrated by physical examination or the ordinary chest film. This fact serves to emphasize the necessity for altering the usual methods of roentgen diagnosis to detect small pleural effusions. This has been ably done by Rigler (3, 4), who showed that all pleural transudates and most pleural exudates, especially those of recent origin and non-purulent character, shift markedly as the position of the patient is changed. By placing the patient in a variety of positions for roentgenographic study, one can take advantage of this fact to detect small pleural effusions and differentiate thickened pleura from fluid. Thus, when a small pleural effusion is suspected because of clouding of the costophrenic sinus, a postero-anterior roentgenogram with the patient in the lateral decubitus position will often disclose a dense or hazy shadow occupying the inferior costal gutter, which in this position is the most dependent portion. This position, therefore, will often reveal the presence of fluid that is concealed by the liver and diaphragm when the patient is erect. When the patient is supine, the characteristic appearance of fluid is no longer present but the involved hemithorax becomes cloudy instead. The use of the Trendelenburg position is also of value, for in this position the pleural fluid shifts to the upper part of the hemithorax, permitting visualization of the part of the lung which is obscured by the fluid when the patient is upright. When no change in the position of the fluid is seen, it is

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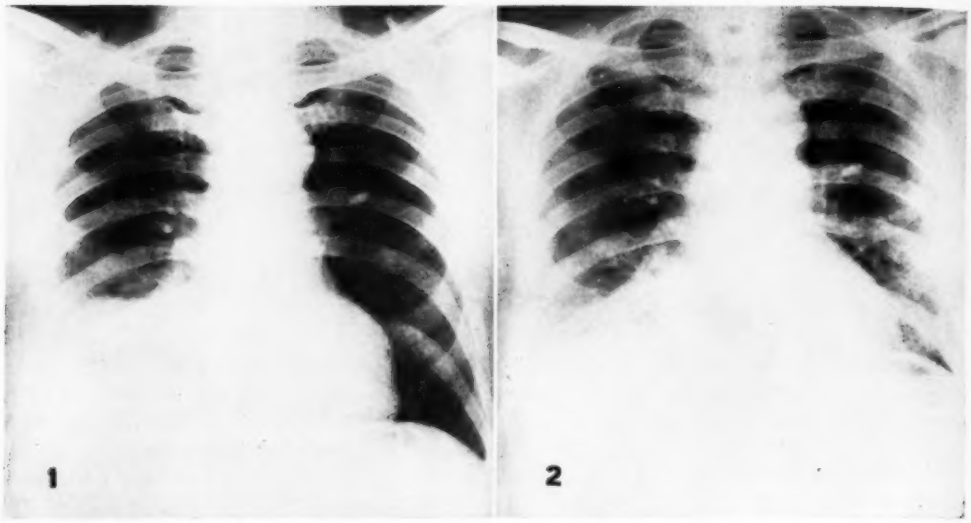


Figs. 1 and 2. Case of tuberculous empyema in which the upper surface of the fluid is rather flat. Figure 2 shows the appearance after removal of the fluid.

usually a purulent exudate or a non-purulent exudate which has been present for a considerable period, allowing the development of adhesions and partial encapsulation (4).

The concavity of the upper margin of pleural fluid is due to a combination of forces (2, 5). Hydrostatic pressure or the pressure of the fluid itself is responsible for its gravitation to the most dependent part of the pleural cavity, resulting in compression of the lung especially at the periphery, where its elasticity is greatest. The lung is partly submerged in the fluid but for the most part floats on the surface. The hilar region, because it is relatively rigid and fixed, is not compressed as much as the periphery. With greater accumulation of fluid, however, the whole lung may be compressed along the mediastinal border. Gravity does not account for the higher position of the fluid at the costal portion of the chest. The normal negative intrapleural pressure, which is maintained by the elastic retractility of the lung, is mainly responsible for this characteristic contour (2, 5, 6). The elastic retractility is greatest peripherally and diminishes as the hilum is approached. Fluid is demonstrable in the costophrenic angle first, because that portion of the lung is the most peripheral and therefore is the site of the greatest retractility. With an increase in the amount of fluid, there is extension along the periphery, where retractility is high.

Closely akin to elastic retractility and partly responsible for the characteristic curve in simple pleural effusions is the cohesive force existing between the pleural surfaces. This cohesion results in capillarity and acts wherever two layers of the pleura are in close proximity. Thus, this force also operates in the interlobar spaces and in the pleural space situated between the parietal mediastinal pleura and the lung. Elastic retractility plays a minor role in this latter location, because of its proximity to the hilum.

The character of the fluid may contribute to the contour assumed by a pleural effusion. It has been observed (6) that transudates are more likely to be atypically distributed than exudates.

We are concerned here with free fluids in the pleural cavity in the absence of pneumothorax. When air as well as fluid is present, the surface of the fluid is flat, as gravity is the only operating force.

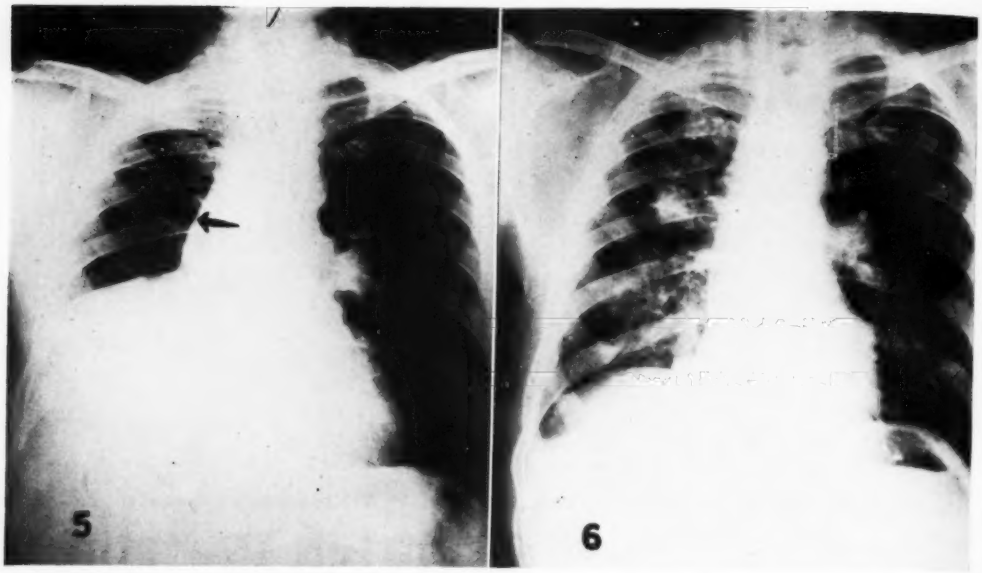
Figs. 3 and 4. Right tuberculous pleurisy with effusion showing flat upper surface. Figure 4 shows the appearance after withdrawal of the fluid.

Since the visceral and parietal pleuras are not in close contact in the presence of pneumothorax, capillarity is no longer effective and, because of the collapse of the lung, retractility is eliminated.

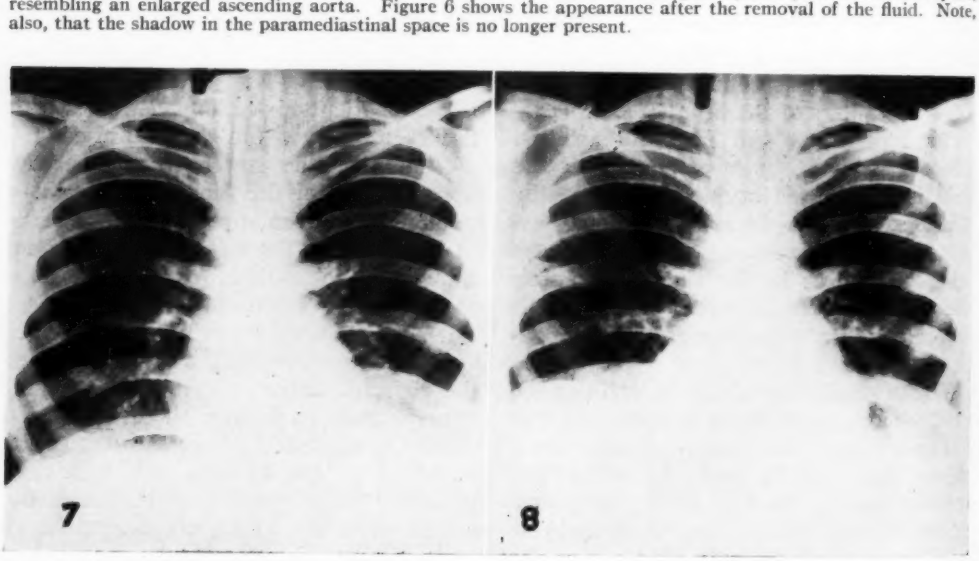
The diagnosis of the usual case of pleurisy with effusion can be made readily. There are cases, however, in which the fluid is distributed atypically. Since the effusion occupies bizarre positions, assumes unusual contours, and simulates other structures in the thorax, diagnosis becomes difficult. It is the purpose of this report to discuss these unusual accumulations of pleural fluid.

The upper surface of the pleural effusion in the absence of pneumothorax may be flat or even convex. It has been our experience that in the cases in which this contour is seen the fluid is very often exudative, with a high specific gravity and protein content. This is in contrast to the opinion expressed by Rigler (6) that the effusions which present an unusual distribution are usually transudative. A case of tuberculous empyema is shown in Figure 1 in which the upper surface of the fluid is rather flat. Figure 2 shows the same case after the fluid had been removed. Another example of the flat surface assumed by a pleural effusion is seen in Figure 3, from a case of tuberculous pleurisy with effusion. Figure 4 shows the same case after the withdrawal of fluid. X-ray films and fluoroscopic examination of these patients in various views failed to disclose any air within the pleural cavity.

When the fluid surface is convex, the interpretation of the roentgenogram may be difficult and the appearance may simulate elevation of the diaphragm, abnormal diaphragmatic outline, or even a subphrenic space infection. In Figure 5 a homogeneous density with a convex upper border is seen overlying the lower half of the right hemithorax. An elevated diaphragm such as is seen in a subphrenic abscess is suggested. Fluoroscopically the upper border moved very little with each phase of the respiratory cycle, but in the supine view the entire right hemithorax became diffusely cloudy and the right leaf of the diaphragm could be seen clearly in its normal position. Figure 6 shows the same case after 1,500 c.c. of clear straw-colored fluid was withdrawn. Another case illustrating this type of pleural effusion is seen in Figure 7. The convex upper border suggests that the left diaphragm is elevated and that the slight haziness above



Figs. 5 and 6. Pleurisy with effusion, in which the upper border of the effusion is convex and simulates an elevated diaphragm. The arrow points to an accumulation of fluid in the right superior paramediastinal space, resembling an enlarged ascending aorta. Figure 6 shows the appearance after the removal of the fluid. Note, also, that the shadow in the paramediastinal space is no longer present.



Figs. 7 and 8. Left pleural effusion with convex upper border. Figure 8 shows the appearance after the removal of the pleural fluid. There continues to be a pleural reaction above the left leaflet of the diaphragm.

this density represents a pleural reaction, fluid, or possibly pulmonary compression. Figure 8, taken after the removal of 300 c.c. of clear amber fluid, continues to show a pleural reaction above the left diaphragm.

In our experience, the most common atypical pleural effusion is that in which the fluid accumulates in the paramediastinal pleural space. The collection of fluid is localized between the parietal

pleura, which forms the lateral boundary of the mediastinum, and the adjacent visceral pleura. Above the root of the lung the paramediastinal pleural space extends uninterruptedly from the sternum to the vertebral column, while below the root of the lung the pulmonary ligament, which extends from the hilum to the diaphragm, divides the space into an anterior and posterior division. Figure 9 shows diagrammatically the most common forms of paramediastinal pleural effusions. An anterior paramediastinal pleural effusion is seen roentgenographically as an opacity having a well defined margin parallel with the right or the left border of the heart, giving the impression that the cardiac margin is reduplicated. This type of pleural effusion is often described as ribbon-shaped. The cardiophrenic angle may or may not contain fluid. Figure 10 shows the appearance of an anterior paramediastinal pleural effusion on the left suggesting an increase in the transverse diameter of the heart. After removal of 1,500 c.c. of clear fluid the actual size of the heart can be seen (Fig. 11). The volume of fluid in such paramediastinal pleural effusions is often surprisingly large. Figure 12 shows a right anterior paramediastinal pleural effusion. The large rounded mass

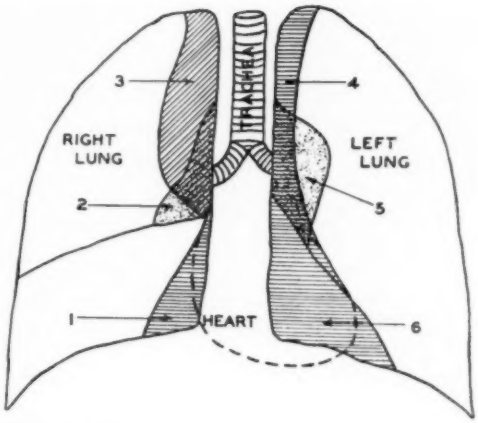
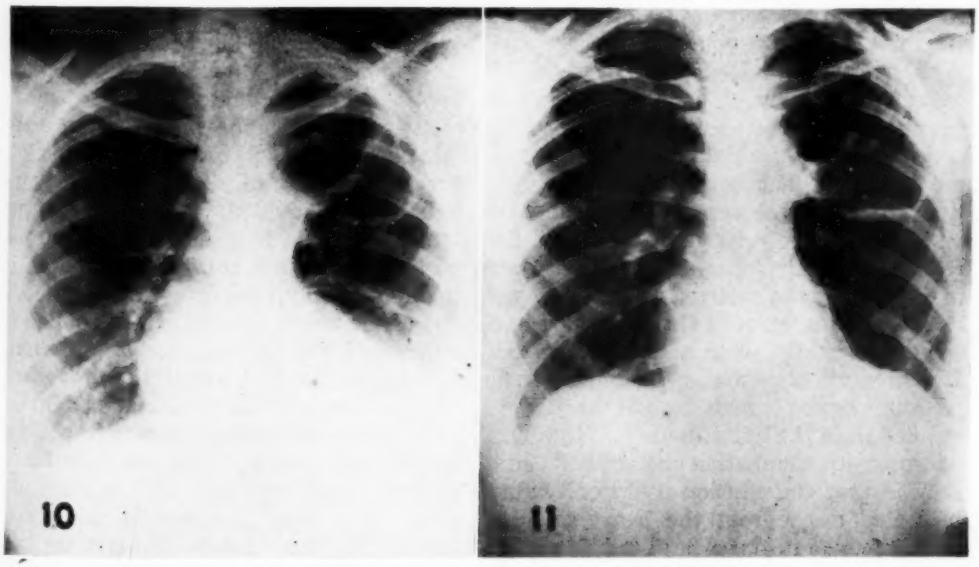


Fig. 9. Diagrammatic representation of the most common forms of paramediastinal pleural effusions (adapted from Harvey, 11). 1. Right inferior paramediastinal effusion. 2. Right parahilar paramediastinal pleural effusion. 3. Right superior paramediastinal pleural effusion. 4. Left superior paramediastinal pleural effusion. 5. Left parahilar paramediastinal pleural effusion. 6. Left inferior paramediastinal pleural effusion.



Figs. 10 and 11. Anterior paramediastinal pleural effusion on the left, suggesting an increase in the transverse diameter of the heart. There is also extensive cavitation in the left upper lobe. Figure 11 was made after the removal of 1,500 c.c. of fluid. The actual size of the heart can be seen.

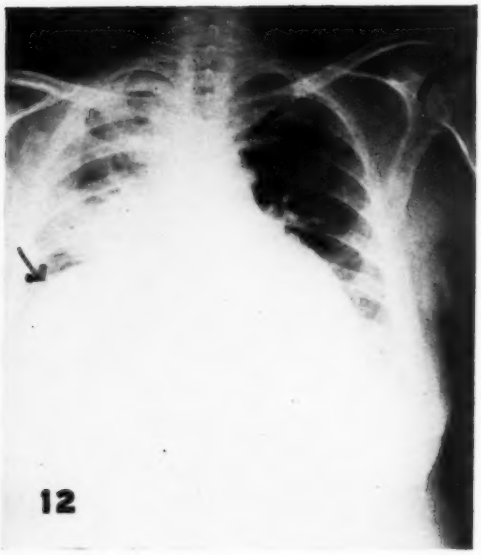


Fig. 12. The arrow points to a large round mass continuous with the heart shadow, resembling right-sided cardiac enlargement, but actually representing a right anterior paramediastinal pleural effusion.

continuous with the heart shadow resembles an enlarged right auricle. The physical signs in this type of effusion are located anteriorly. Dullness continuous with that of the heart is usually present, and over the area of dullness the usual signs of pleural effusion are in evidence.

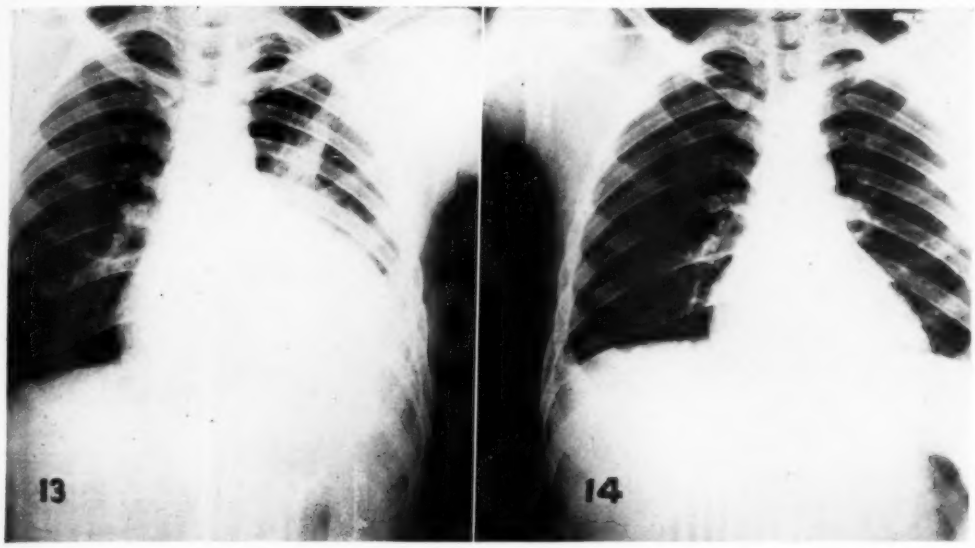
Posterior paramediastinal pleural effusions present ribbon-shaped shadows running parallel to the right or left borders of the vertebral column or triangular opacities with the base resting upon the diaphragm and the apex of the triangle directed toward the hilum. The case shown in Figure 13 was referred to the hospital because it was believed that the heart had enlarged rapidly over a six-day period. It represents a posterior paramediastinal pleural effusion which cannot be distinguished from the heart shadow. However, fluoroscopic examination and oblique views showed that the effusion could be readily projected away from the heart shadow. After removal of the pleural fluid, the true size of the heart could be seen (Fig. 14). Physical signs may be absent, but when the effusion extends to the chest wall, the signs of pleural effusion are present lateral to the vertebral column.

In Figure 5 the arrow points to a collection of fluid in the paramediastinal pleural space, extending above the root of the lung. This shadow simulates a mediastinal tumor or an enlarged ascending aorta. After the removal of the fluid, this shadow is no longer present (Fig. 6).

In most cases the heart and adjacent paramediastinal pleural fluid are of different densities and therefore readily distinguishable (Figs. 15 and 16). At times, however, the pleural fluid and heart are almost identically opaque and it may be difficult to determine where the lateral border of the heart ends and the pleural effusion begins. It is this latter type of effusion that so closely simulates cardiac enlargement or encapsulated pericardial effusion (Figs. 10, 12, and 13). Oblique and lateral views combined with a fluoroscopic examination will readily disclose the true nature of the pathological process.

In all of our cases the paramediastinal pleural effusions were of tuberculous origin. Any of the many causes of pleural effusion may, however, result in an accumulation of fluid in the paramediastinal pleural cavity.

Clinical recognition of fluid in the interlobar spaces is difficult. A thorough knowledge of the anatomical location and roentgenological appearance of the interlobar fissures is important in the evaluation of this type of effusion. Briefly, the left lung is divided into an upper and lower lobe by a long fissure which begins posteriorly some 6 cm. below the apex of the lung at about the level of the vertebral end of the fourth rib and extends obliquely downward and forward, ending anteriorly at the costochondral articulation of the sixth rib, which is near the cardiac apex. On the right, the long fissure is similar in position to the long fissure of the left lung but is slightly more vertical. It separates the lower lobe from the middle and upper lobes. The short fissure begins in the long fissure in the axillary region at the level of the fourth rib and extends horizontally forward to the costochondral articulation



Figs. 13 and 14. Posterior paramediastinal pleural effusion on the left, giving the impression of cardiac enlargement. After the removal of the pleural fluid (Fig. 14) the true size of the heart can be seen.

of that rib. It separates the upper lobe from the middle lobe. These fissures extend to the hilum of the lung.

We are not considering here encapsulated interlobar effusions that remain confined to the interlobar space regardless of the effect of gravity, but rather fluid that is free in the general pleural cavity but extends into the interlobar fissure. At times, the presence of the effusion in the interlobar fissure can be seen only when the patient is placed in certain positions.

The short interlobar fissure is more commonly the site of interlobar pleural effusions than the long fissures. In Figure 17 the pleural effusion occupies the costophrenic angle and extends upward along the axillary border to the short interlobar fissure, where the narrow horizontal band represents an accumulation of fluid at this site. When only a small amount of fluid is present in the pleural cavity, it does not rise as high as the short fissure with the patient erect. When the horizontal position is assumed, however, the fluid reaches a higher point and may be drawn into one of the interlobar fissures by the force of capillarity existing there. This is even more likely to occur in the lateral decubitus position, in which instance the fluid occupies the inferior costal gutter and readily enters one of the interlobar spaces. When the effusion is large enough, as in the case seen in Figure 17, the fluid reaches and enters the interlobar fissure even when the patient is upright. In this event the interlobar effusion becomes considerably wider and even ovoid in the horizontal or lateral decubitus position (8).

The x-ray appearance of the interlobar portion of the pleural effusion is determined by the course of the interlobar fissure (Figs. 17 and 18). In any event, the density is horizontal, vertical, or oblique; it extends from the hilum, and appears at different levels of the lung fields depending upon the part of the fissure involved. The size and shape of the interlobar effusion vary according to the volume of the fluid and the compressibility of the adjacent lobes (8). The smaller interlobar effusions are narrow and band-shaped, whereas the larger ones are ovoid or circular. In the lateral position the shadows have clearly defined upper and lower borders which are either straight or convex.

This type of interlobar effusion is most commonly of tuberculous or pneumonitic

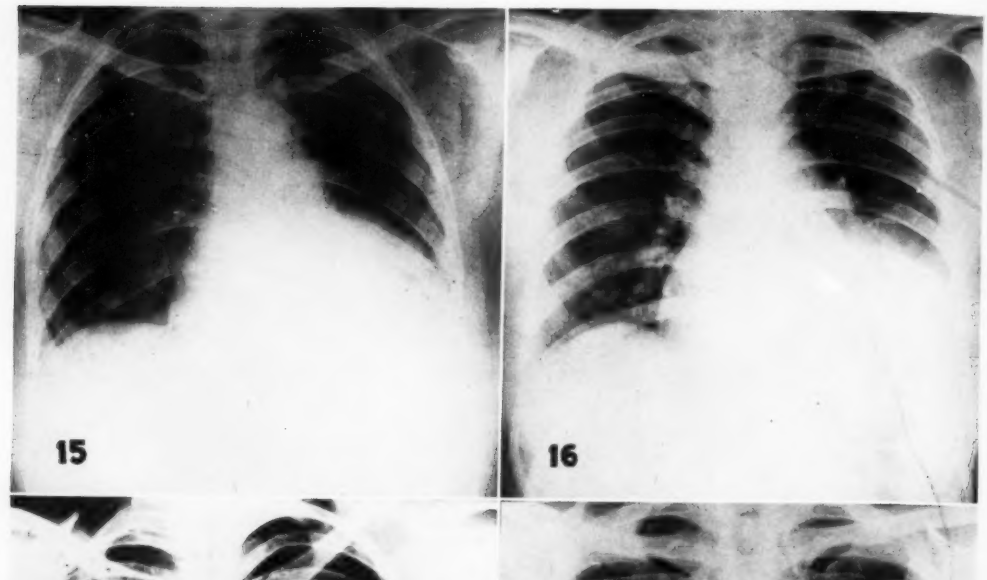


Fig. 15. Ribbon-shaped shadow along the left border of the heart, representing a paramediastinal pleural effusion. The heart and paramediastinal pleural effusion are of different densities and therefore readily distinguishable.
Fig. 16. Pleural effusion obliterating the left costophrenic angle and extending along the left border of the heart in the left paramediastinal pleural space.
Fig. 17. Pleural effusion occupying the right costophrenic angle and extending upward along the axillary border to the short interlobar fissure, where the narrow horizontal band represents an accumulation of fluid at this site.
Fig. 18. Pleural effusion occupying the right costophrenic angle and extending into the interlobar fissure between the lower and middle lobes.

origin but may be seen occasionally in cases of congestive heart failure.

Many reasons are offered for the unusual appearance of atypical pleural effusions. Surely, any factor altering the retractility of the lung will affect the contour of the pleural fluid. Thus, pulmonary fibrosis, consolidation, or edema, by reducing the retractile power of the lung, allows gravity to exert the greatest influence, and therefore the fluid level may not be concave (6). Changes in the character of the pleura produced by inflammation or adhesions will affect the shape of the fluid considerably.

Obliteration of portions of the pleural cavity may determine the localization of pleural effusion. In the interesting case of pleural effusion simulating elevation of the diaphragm reported by Yater and Rodis (9), a satisfactory explanation for the unusual appearance of the tuberculous pleural effusion was not obtained from the necropsy. It was concluded that the fluid was encapsulated above the diaphragm by thin fibrinous adhesions but escaped rather freely as the position of the patient was changed. The effect of adhesions on the contour of pleural effusions is considerable. Pleural adhesions fortuitously situated may confine fluid in a strange way.

Korol and Scott (10) have attempted to explain the convex upper border occasionally seen with pleural effusions on the basis of a hydropneumothorax, with the air pocket obscured by a partly expanded and adherent lung. They state that the lateral decubitus position with the patient lying on the normal side will demonstrate the concealed air bubble above the fluid level. While this explanation may occasionally obtain, it is not satisfactory in most cases, for the patients having pleural effusions of unusual type are usually examined in a variety of positions, without any pneumothorax being demonstrated. Furthermore, in our cases there was no evidence of spontaneous pneumothorax nor was the accidental introduction of air into the pleural cavity possible, since thoracentesis had not been performed before the x-ray films were made.

Although there are numerous factors which influence the variation in appearance of pleural effusions, it must be admitted that in many cases no satisfactory explanation is apparent.

SUMMARY

A review of the cases that have been presented demonstrates the unusual positions and contours that may be assumed by pleural effusions, as well as the diagnostic confusion that may arise. It is to be emphasized that fluoroscopic and roentgenographic examinations in a variety of positions are mandatory for proper evaluation of the process.

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Radon Ointment Treatment of Irradiation Ulcers[1]

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LARGE IRRADIATION ulcers with accompanying necrosis, infection, and sloughing, and often eventual malignant changes, have long been a serious therapeutic problem. Treatments designed for ordinary deep burns are not effective; the ulceration produced by atrophy of the skin and vascular obliteration is extremely indolent. Probably the only effective treatment has been excision and skin grafting. Because of the usual infection and malignant changes, along with the reduced blood supply, satisfactory results have not been frequent.

Fortunately, fewer of these extensive ulcerations are now seen than formerly. Better understanding of the biologic effects of irradiation and the more accurate determination of dosage have contributed to this improvement. The tissues of some patients, however, prove abnormally sensitive to roentgen and gamma rays. Furthermore, to arrest many deep-seated cancers, irradiation has to be pushed to the limits of tissue tolerance. Instances of slowly developing, severe injuries from irradiation given many years ago are seen frequently enough to make the problem an urgent one.

Since 1930, Uhlmann (5) has advocated the application of petrolatum impregnated with radon. He has expressed the belief that the mechanism involved is the superficial effect of alpha particles, previously never used therapeutically. In ordinary radium therapy, radium or its decay products are always enclosed in glass or metal, or both, preventing the egress of alpha radiation. Although there seems to be no logical reason to employ radon to treat an injury produced by radium or roentgen rays, yet radon absorbed in petrolatum is a rich source of a type of radiation—alpha particles—not involved in the production of these injuries.

We have treated two patients, the first two to receive this form of therapy at the Mayo Clinic. In each case the condition was unusual; ulceration of many years' standing had undergone malignant change, and all methods of treatment hitherto tried had been employed without avail. Both patients were treated by the application of petrolatum that contained radon, in an attempt to heal the ulcers, and by highly filtered radium to control the malignant changes.

REPORT OF CASES

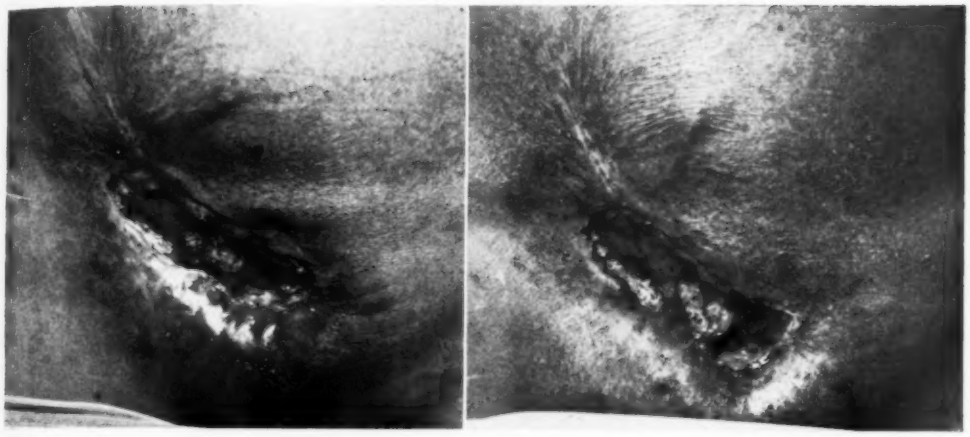
CASE 1: A girl, aged eighteen years, first registered at the clinic in 1919 with an open draining wound in the right lower quadrant of the abdomen. She stated that in November 1917, because of severe pain in the right lower abdominal quadrant, appendectomy had been performed with removal of a sound appendix. A few months later, the wound broke down and remained open and draining. At another operation, performed in April 1919, necrotic tissue was excised, but closure of the wound was not achieved. Low-voltage roentgen treatment had been given in an effort to obtain healing. This seemed to relieve pain, but complete healing did not occur. The patient exhibited a large element of hysteria, and it was thought that the wound had been kept open by her interference. A diagnosis of traumatic ulceration and hysteria was made.

In September 1926, the patient returned. She had married during the previous month. The wound was still open and draining in spite of radium treatment which, she said, she had received three years previously. She had been taking large doses of morphine during the past five years.

In 1930, the ulcer had temporarily healed except for scabbing, and the morphinism had been controlled.

The patient's next visit was in November 1942. A large draining ulcer was present over McBurney's

[1] Presented at the Joint Meeting of the American Roentgen Ray Society and the Radiological Society of North America, Chicago, Ill., Sept. 24–29, 1944.



Figs. 1 and 2. Traumatic ulcer with irradiation damage, before treatment with radon in petrolatum (December 1943) and after one course of radon ointment (March 1944). Clinically carcinoma was present.

region. Biopsy showed inflammatory tissue with epithelial hyperplasia. The condition was regarded as traumatic and factitial dermatitis with probable irradiation injury superimposed.

In spite of the negative biopsy, the clinical diagnosis was low-grade carcinoma with metastasis, based on the appearance of the ulcer and the presence of enlarged nodes in the inguinal and femoral regions. The ulcer was deep and measured 9 × 3 cm. Its base was necrotic, with a foul discharge; the edge was raised and indurated, with infiltration extending into the surrounding skin. Conservative treatment with potassium permanganate dressings was prescribed.

On the patient's return in September 1943, the ulcer still drained freely. The odor was characteristic of a malignant neoplasm and the nodes in both groins had enlarged and become fixed. The right femoral nodes were also involved. Radium treatment to the metastatic nodes was administered at this visit.

By December 1943, the inguinal nodes were unchanged, and enlarged femoral nodes made walking difficult. Radium treatment of the nodes was again carried out.

The abdominal ulcer was painful and draining (Fig. 1). Radon ointment therapy was employed once a week for nine weeks. The ointment was freshly prepared in our physics laboratory. The applications varied in strength from 0.031 to 0.05 mc. of radon per cubic centimeter of petrolatum, averaging 0.04 mc. per cubic centimeter; approximately 12 c.c. was applied at each dressing. The pain ceased with the second or third application and drainage diminished.

In March 1944, the malignant infiltration had spread through the suprapubic region and the upper part of the vulva; therefore, further radium treatment was applied to these areas at that time and again in June.

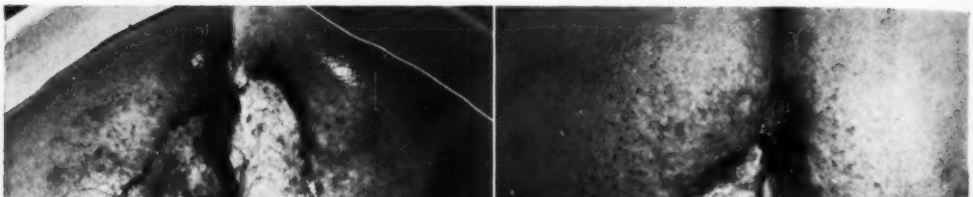
During July and August, a second series of weekly applications of radon ointment was given, the strength of the ointment varying from 0.08 to 0.10 mc. of radon per cubic centimeter of petrolatum.

Figure 2 shows some improvement. The ulcer measures 7.5 × 3 cm. and is decidedly shallower than before. Formation of epithelium has occurred along the top and sides. At this time there was no pain whatever and less drainage than before.

CASE 2: In December 1942, a man of seventy-three years registered at the clinic. Besides prostatic hypertrophy and mild diabetes mellitus, his main complaint was a large perianal ulcerated tumor, which had been present for many months. He stated that he had been given roentgen therapy in 1925 for anal pruritus, which had healed perfectly under treatment. By 1930, a reddened area appeared just above the anus, which became indurated in 1933. By 1936, he noted scaling and increasing soreness, and gradually the induration spread until it almost completely covered the anus and produced difficulty with defecation.

Examination showed an indurated, ulcerated lesion, measuring 2 × 3 cm., in the posterior margin of the anus externally, with a large area of actinodermatitis extending over both buttocks. Many lotions had been applied without improvement.

In December 1942, transurethral prostatic resection was performed, and in February 1943, an attempt was made to cure the perianal lesion by surgery. Palliative excision of the ulcerated lesion was performed and tissue was taken for biopsy from three widely separated parts of the ulcer; all these biopsies showed squamous-cell epithelioma, grade 2 (Broders). Removal of most of the neoplastic tissue was carried out, but some of it could not be removed because complete excision would involve cutting into the rectal wall. The malignant ulcer was heavily treated with radium in March



Figs. 3 and 4. Perianal lesion before application of radon ointment (July 21, 1943) and after application of radon ointment (May 19, 1944).

1943. This treatment has served to arrest the malignant lesion to date. However, the large open ulcer, as painful as the previous lesion, remained.

Many therapeutic measures were employed in the attempt to heal this huge roentgen ulcer. Extract of Aloe vera was applied, as well as ointments containing gramicidin, sulfonamide compounds, and aluminum acetate washes. Improvement did not follow any of these measures.

Radon ointment therapy was started July 27, 1943, and was continued once a week through Oct. 19, thirteen treatments in all. The dose ranged from 0.03 mc. to 0.07 mc. per cubic centimeter of petrolatum, with an average of 0.05 mc. per cubic centimeter. Approximately 12 c.c. of petrolatum was used at each application.

The original lesion is shown in Figure 3, the photograph having been taken July 21, 1943. The patient felt more comfortable after the first two or three applications than he had felt before, and a gradual clearing up of the sloughing, indolent lesion became evident. Eventually, complete epithelization occurred, as shown in Figure 4, a photograph taken May 19, 1944. Pain has disappeared completely and the patient is now able to walk, to lie on his back, and even to sit down with some comfort.

HYPOTHESES REGARDING THE EFFECTS OF RADON OINTMENT

Four possibilities are suggested to explain how the radon in petrolatum produces a beneficial effect on the tissue. These are as follows: (1) the action of the radiations on the tissue itself; (2) the action of the radiations on the petrolatum; (3) the simultaneous action of the irradiations and the petrolatum; (4) the action of the disintegration products of radon on tissue.

Action of Radiations on the Tissue: The tissue dose from gamma and beta radiation is probably too small to have any significant effect. The dose from the alpha radiation would be very great but would be confined to the tissue within a radius of about 0.1 mm. of the disintegrating radioactive atoms. If any beneficial effects are produced because of alpha irradiation, then it is essential that the ointment come into contact with the tissue so that the alpha particles can penetrate into it. The depth of tissue affected is undoubtedly increased because of penetration of petrolatum containing radioactive atoms and by absorption of radon by the tissue. There is also the possibility of a beneficial effect being produced as a result of the irradiation of the tissue fluids as well as the cells.

Although it generally has been thought that any of the common types of radiation is harmful rather than beneficial to irradiation injuries, little is known about effects produced by alpha radiation. It has been assumed that its action, except for the fact that it is very concentrated, is essentially the same as that of other types of radiation. This greater concentration of absorbed energy may start a series of biologic reactions which differ in some respects from the series of reactions started by other types of radiation. Erythema similar to that produced by roentgen or gamma rays was produced by the acci-

dental irradiation of the back of a hand by alpha rays from a cyclotron a few years ago (2), but this does not necessarily mean that all the other biologic reactions produced by the radiations were similar.

Action of Radiations on Petrolatum: The intense irradiation received by the petrolatum must produce some chemical and physical changes, and it is possible that some of these changes might make it a more beneficial therapeutic agent. A commercial brand of petrolatum which is irradiated with ultraviolet rays is supposed to be more beneficial for some purposes than ordinary petrolatum. Stenstrom and Vigness (4) have shown that irradiation of heavy mineral oil by roentgen, beta, and ultraviolet rays causes it to spread out over a larger area when it is placed on water; a somewhat similar effect might be produced by alpha radiation on petrolatum. If any therapeutic effect of radon in petrolatum is due to the action of the radiation on the petrolatum, it might be possible to obtain the same result by treating petrolatum with some other types of radiation before it is applied to the lesion. If, however, the changes produced in the petrolatum were unstable, it might be necessary to irradiate the petrolatum during application, in which circumstance alpha radiation might be the radiation of choice since, because of its low penetrating power, it would not affect deeper tissue.

Simultaneous Action of Radiations and Petrolatum: Irradiation produces changes in tissue, such as ionization, changes in cell permeability, and changes in osmotic pressure, and irradiation must produce some physical and chemical changes in petrolatum. Perhaps it is a combination of these effects that is therapeutically beneficial.

Action of Disintegration Products of Radon: In petrolatum containing radon there would be traces of radioactive isotopes of other elements. Until the instant that these radioactive atoms disintegrate, they act chemically and physiologically in the same manner as non-radioactive elements. The amount of these isotopes present in each cubic centimeter of petrolatum would depend on the concentration of the radon, and the amount of one of them (the isotope of lead, RaD) would depend, also, upon the length of time the radon had been in the petrolatum. The order of magnitude of the amounts of these radioactive isotopes in each cubic centimeter of the petrolatum would be 10^{-10} mg. of polonium, 10^{-9} mg. of bismuth, and 10^{-7} mg. of lead. These values seem like insignificant amounts but, when expressed as number of atoms per cubic millimeter, they are, respectively, of the order of 100,000, 1,000,000, and 100,000,000. The presence of these isotopes in the petrolatum on the surface of the lesion would be of little importance, but if the petrolatum penetrates into the tissue, these elements might be available to the tissue; also, the radon which may be absorbed by the tissue would form these isotopes within the tissue as it disintegrated. In recent years it has been realized that traces of eelments may be of great physiologic importance (1), and the added fact that these isotopes are radioactive may make it worth while to consider whether or not their presence is of any significance.

SUMMARY

The first two patients to be treated with radon ointment at the Mayo Clinic presented about as hopeless a prognosis as could be imagined. Both were afflicted with massive ulcers of many years' duration. In the first case the ulcer may have originally been traumatic, with irradiation damage probably superimposed. In the second case, the lesion was assuredly a roentgen ulcer. In each case, a malignant lesion had finally appeared, with distant metastasis in the first instance and localized cancer in the second.

In both cases, radium therapy was employed to arrest the malignant lesion and radon ointment was used to treat the ulcer.

In the first case, results of radon ointment therapy were encouraging; evidence of healing has occurred, but further treatment seems necessary. In the second case,

the more prolonged treatment given has resulted in a complete healing of the ulcer.

Radon ointment therapy must be carefully distinguished from radium therapy. If the effect is due to the alpha particles, it is due to radiation of a quality not employed in radium therapy. It is known that alpha radiation is extremely powerful, and Martland (3) has condemned it as a lethal agent when radon salts are swallowed or injected into the system.

Radon ointment therapy is in an experimental stage. So far, the work of Uhlmann has shown promise. If further work continues to yield good results, we have an important adjunct to irradiation therapy. The present vogue of intensive irradiation to arrest deep-seated malignant lesions may produce many irradiation ulcers in future years. A satisfactory method of treatment of these ulcers will be a tremendous boon.

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DISCUSSION

Erich Uhlmann, M.D. (Chicago, Ill.): I am glad Dr. Fricke has had an opportunity to treat some cases of irradiation injury with radon ointment, as I have been using this for almost sixteen years, with gratifying results. As to the mechanism of the effect, I have been conducting some experimental studies, as yet unpublished. In view of these observations, I do not believe that the vaseline or petrolatum, as such, has anything to do with the effect of the ointment. It is used because of its inertness and because the amount of radiation or radon absorbed by it is large—sixteen times that of water at room temperature. [At this point Dr. Uhlmann showed slides and presented brief histories of two cases treated with radon ointment. He stressed the point that the ointment has no influence on malignant growth. In each of the two cases reported a carcinoma was subsequently removed surgically.]

Lowell S. Goin, M.D. (Los Angeles, Calif.): I was anxious to say something about this paper of Dr. Fricke because in conversation and correspondence I have met with expressions of rather contemptuous doubt as to there being any sense at all to this whole procedure.

I have treated 18 cases of radiation injury with radon ointment, and I wish to say that I merely collected these cases; I didn't cause them! Results have been not 100 per cent but far better than anything I have ever seen. In fact, I have never seen a typical radiation ulcer heal under any other form of therapy. As Dr. Uhlmann says, it doesn't seem to make too much difference whether lanolin or vaseline is used as the vehicle. I have used both.

I should like to give you a brief account of one case. A young woman who had been treated for a presumed malignant melanoma on the lateral aspect of the left knee had a radiation ulcer one year after treatment. The lesion was roughly circular, measuring approximately 10 cm. in diameter, exposing the head of the fibula and the lateral ligaments of the knee. The knee was contracted to forty-five degrees, and the pain was so agonizing that she begged for amputation. She has received either eighteen or nineteen weekly applications of radon in lanolin. At present she has a shallow, painless, slightly ulcerated lesion about the size of the little finger nail, which is dotted with islands of epithelium. The ligaments and head of the fibula are completely covered. I cannot believe that such a result could have been obtained in any other way.

Neither Dr. Fricke in his paper nor Dr. Uhlmann in his discussion has mentioned the technic. It must be followed exactly as outlined in Dr. Uhlmann's original paper.

Lyell C. Kinney, M.D. (San Diego, Calif.): I realize, as Dr. Fricke has said, that this method of treatment is still in the experimental stage and that there are few men in the country who are using it, but such amazing results have been recorded in otherwise hopeless cases that I believe that the investigation should be continued until we can justly appraise it.

A young doctor came into my office in February with a third-degree radiation dermatitis of the index finger. The distal phalanges were covered with a leathery skin that looked like soaked sole leather—moist and entirely free from sensation. At the upper end was a line of demarcation. It looked as though the two distal phalanges of the finger would be lost, and the proximal phalanx was acutely inflamed and exquisitely tender.

The consultant in the case had decided to amputate the finger and, as a last resort, the senior physician in the department asked us to look at it. At the suggestion of Dr. Lowell Goin, we treated the lesion with radon ointment. Twenty applications

were made, following Dr. Uhlmann's technic. I saw the patient this last week. The skin was soft and pliable to the very end of his finger, and there was no pain or tenderness. That young surgeon is very grateful for the conservation of his index finger, and I feel sure that such a result could have been obtained in no other way.

Robert E. Fricke, M.D. (*closing*): I wish to thank the discussants for their interest and for the valuable contributions they have made to the subject. Dr. Uhlmann brought out a very important point; namely, that the radon ointment has no effect on malignant lesions whatever. One has to remember this and treat the malignant lesion separately whenever it is present in these ulcers.

As to what type of lesions to treat with radon ointment rather than operation, I do not think we are yet far enough advanced to make proper selections. The radon ointment therapy takes many weeks and is tedious, but surgical treatment is also a tedious matter. The important thing is, I believe, that there are now two methods of treatment, and if one fails, the other might be tried.

Capsular Osteoma of the Knee Joint. Report of Four Cases[1]

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New York, N. Y.

PRIMARY TUMORS of the knee joint may derive from the meniscal or paramenisical region, from the synovial membrane and villi, from the subpatellar fat pad of Hoffa, from bursae, and from capsular or paracapsular structures. The tumors may be benign or malignant, single or multiple. They may be classified as follows:

A. Benign

1. Chondroma (single); chondromatosis (multiple)
2. Osteoma (single); osteomatosis (multiple)
3. Ganglioma; neuroganglioma
4. Fibroma
5. Xanthoma, solid and cystic forms
6. Chloroma
7. Lipoma and lipophagic granuloma
8. Myxoma
9. Hemangioma and lymphangioma
10. Meniscal cysts
11. Traumatic hypertrophy and sclerosis of Hoffa's fat pad

B. Malignant

1. Synovial epithelioma, mesothelioma, endothelioma and perithelioma
2. Sarcoma: fibrosarcoma, spindle-cell sarcoma, round-cell sarcoma, giant-cell sarcoma
3. Myxochondrosarcoma and polymorphous sarcoma

Tumors of the joint capsule range with the tumors of the tendon sheaths and of the serous membranes and because of their topographical location and histologic structure present a similar and fairly characteristic pathological pattern and physiological behavior. The synovial membrane and the paracapsular tissues (16) are the site of origin. The largest group consists of cartilaginous tumors due either to faulty mesenchymal differentiation or to traumatic detachment of chondroblastic elements with subsequent synovial implantation (3). Chondroma and chondromatosis may follow osteochondritis dissecans, chronic hypertrophic osteoarthritis, neurogenic arthropathies, hemophilia, aseptic necrosis, ochronotic alkaptonuria, postinfectious joint destruction, meniscal tears, and other cartilaginous damage (4, 13).

While the roentgen appearance of joint chondromatosis is well known, reports of osteoma of the knee joint have been scarce. Kienböck in 1924 (8) reported an intracapsular osteoma of the knee with a correct preoperative diagnosis. He was able to collect from the literature similar lesions reported during the roentgen era by Marchand in 1917, M. B. Schmidt in 1918, Trapp in 1896, Rumpel in 1908, and by Baetjer and Waters (1) in 1921. The roentgen diagnoses in these cases were, variously, exostosis of tibia, fibula, and patella, osteogenic tumor, and accessory patella. By carefully redrawing the radiographic images, however, and by studying the operative reports, Kienböck was able to identify the roentgenologic and surgical findings with those in his own case. Several more examples were seen by Kienböck up to 1934, and in his standard work on "The Roentgen Diagnosis of Bone and Joint Diseases" he concludes that osteoma occurs rarely in other joints, such as the elbow, hip, shoulder, and ankle, that the size of the tumor is hardly in proportion to the low degree of functional impairment, that incarceration does not occur, and that the blood vessels and surrounding skeletal parts are never invaded. Osteoma of the knee joint is a new growth of synovial origin and it therefore belongs to the group of mesenchymal hyperplasias or dysplasias.

[1] From the Department of Radiology of the New York Hospital, New York, N. Y. Accepted for publication n November 1944.

Fig. 1. Case I. Patella-shaped shadow of osteoma beneath the patella. Adjoining bone surfaces are smooth.

Since the establishment of the clinical, roentgenologic, and pathologic characteristics by Kienböck, osteoma of the knee joint has been reported by Böhm (2), Hammer (6), Jerusalem (7), Laurence (10), Razemon and Bizard (12), Robillard (13), Schnaberth (14), Simon (15), and Weiss and Löwenstein (17). Only two cases have been found in the American literature (Baetjer and Waters; Robillard). Four more cases of this rare joint condition have been seen by the author.

CASE REPORTS

CASE I: This observation dates back over fifteen years and a detailed history is not available. The patient was a middle-aged housewife with pain and some limitation of motion in the left knee. She recalled many years of floor sweeping while a girl. A hard mass could be felt somewhat lateral to and below the left patella.

Roentgen examination revealed a well delineated bony tumor with small areas of increased radiolucency. The outlines of the tibial surface were irregular and hazy and there was slight subchondral demineralization (Fig. 1). The specimen removed by operation was a solid bony tumor with some fibrous, cartilaginous, and fatty tissues on the surface. Hoffa's fat pad was completely atrophied.

CASE II: A white housewife, aged 51, was admitted for pain and stiffness of the legs and knees, especially of the left knee. The pain was burning in type and worse at rest than after a period of limbering up. The knee became progressively more stiff and could not be flexed beyond a right angle. The joint was swollen, and palpation yielded a solid sense of fullness but no evidence of fluid.

Roentgenograms revealed a large, well delineated shadow of bony density just beneath the patella, overlying the opposing surfaces of the lateral femoral and tibial condyles. The shadow was well separated from the adjoining bones, and the joint surfaces were negative for evidence of invasion, erosion, or decalcification (Fig. 2). At operation a tumor, $5 \times 4 \times 2.5$ cm. was easily removed from the retrofascial cavity. The fat pad was completely gone and the tumor had displaced the patellar ligament in a lateral direction. The specimen (Fig. 3) consisted of a hard tumor with a uniformly smooth and unbroken surface, containing cartilage. Microscopic examination showed bone with fat marrow and many small blood vessels. Along the edge were many osteoblasts which were actively laying down bone. The patient made an uneventful recovery and regained almost complete free mobility of the joint (Fig. 4).

CASE III: An Italian born male barber, aged 47, had noticed a hard lump on the left knee for ten months, with pain after sitting in one position. There was no history of a single injury, but the patient admitted having hit the left knee many times on the barber chair since boyhood. The knee became swollen on the anterior aspect but it was not tender to palpation except when in flexion or extension. There was an ovoid hard mass just below and medially to the left patella, not tender and freely movable apart from the patella itself. There was no limitation of motion of the knee joint.

Roentgenograms revealed a well delineated ovoid bony shadow beneath the patella, by which it was in parts overlapped. There was normal bony trabeculation, with some rarefied areas, in this shadow. Slight osteoarthritic changes of the joint surfaces were present (Fig. 5). At operation a bony tumor $4 \times 4 \times 2$ cm., was easily removed, leaving the joint capsule intact. The tumor was in no way directly connected with the patella, and the fat pad was well preserved.

The tumor was covered with fibrous tissue and the exterior was not unlike cartilage. Microscopic sections revealed a trabeculated pattern of highly vascularized bone with hyaline cartilage and with thick fibrous tissue on the surface. After an uneventful postoperative course, the patient recovered almost normal mobility of the knee.

Figs. 2–4. Case II. Figure 2 shows the knee before operation; Figure 3 the operative specimen; Figure 4 the postoperative appearance

CASE IV: A male teacher, aged 27 years, stated that, while he was still a boy, his left knee suddenly gave way. A diagnosis of a torn cartilage was made, and roentgenograms were reported as negative for bone changes. Several weeks later a second injury occurred, apparently without any further damage to the knee. During the ensuing years a hard lump below the knee cap began to form and the knee was always weaker than the opposite one. Following a recent sprain of the left knee, the patient noticed swelling and slight pain for a short time.

Roentgenograms revealed a large, well delineated shadow of bony density in the lateral anterior joint space, overlapping the anterior tibial surface. The bony surfaces of tibia and fibula were smooth. The patella was dislocated in an upward direction (Fig. 6). The patient refused surgical intervention because of the little trouble caused by the lesion. He was seen over one year after the first examination and was still unwilling to submit to an operation, because, as he said, the knee did not bother him.

COMMENT

These four cases are almost identical in history, clinical course, and roentgen findings. In three cases the tumor was removed by operation and the true osteomatous character of the lesion was proved by macroscopic and microscopic examination. In every instance the left knee was

involved and the lesion was demonstrable as a well delineated patella-shaped shadow of bony density and pattern. This shadow was free from any osseous connection with neighboring bones and there was little evidence of secondary changes of the adjoining bone surfaces. In one case the patella was displaced cephalad. In each instance the tumor was located just beneath the patella, either slightly medially or laterally, overlapping somewhat the anterior surface of the tibial head. The degree of restriction of joint motion was slight over many years and in the operated cases pains and some limitation of motion had increased only lately. Three patients admitted repeated traumatism dating back to the years of adolescence: the housewife complaining of pain as the result of many years of floor sweeping in girlhood, the barber stating that he had frequently hit his knee against the barber chair, and the teacher giving a history of repeated injuries in boyhood.

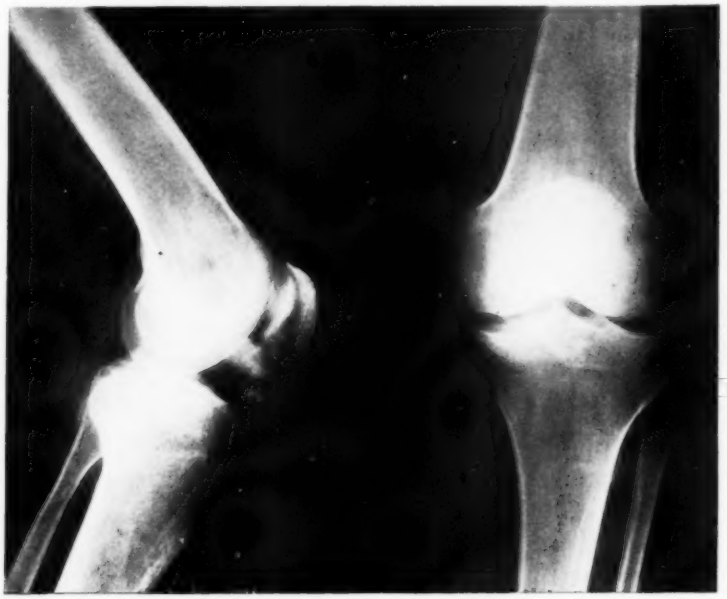

Fig. 5. Case III. Ovoid bony shadow beneath the patella. Minimal osteoarthritis of tibial joint surface.

In the three operated cases the tumor could be easily removed and was free of any strong tissue connection or bony bridges. Microscopic examination showed true bone tumors containing highly organized bone tissue and evidence of a living organized body with blood vessels, fibrous and villous tissue about the surface, and some cartilage. At the free tumor surface facing the joint space, near the posterior capsular layers, the amount of fat, cartilage, and fibrous tissue was greatest. All tissues were in normal and active proliferation and failed to show evidence of malignant change. Only in Case III were there mild chronic hypertrophic osteoarthritic changes. In two cases there were some indentation and shallow surface depression of the anterior tibial surface, apparently due to long-standing pressure.

The four cases show the roentgen characteristics outlined by Kienböck. Roentgenograms of osteoma of the knee joint present a well delineated shadow of bony density with cancellous and sometimes spongy pattern, with here and there small irregular areas of increased radiolucency.

The latter areas contain roentgen-negative tissues, such as fibrous, synovial, fatty, and cartilaginous elements. There is a more or less continuous dense outer shell, and the shape of the shadow may be that of an hourglass, an Indian club, an egg, or of the patella. The shadow usually occupies a large part of the anterior compartment of the knee joint and more often lies laterally than medially. The position may vary, however, and similar tumors have been observed anywhere within the upper and lower, the anterior and posterior portions of the joint capsule. The tumor can always be separated from the adjoining skeletal parts. Herein lies the distinction between joint osteoma and osteogenic tumors, such as exostosis, osteochondroma, and giant-cell tumor of the bone.

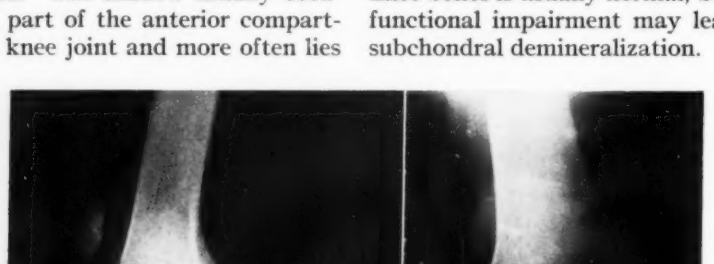

Fig. 6. Case IV. Transversely placed patella-shaped bone shadow at level of the joint space. The patella is dislocated upward.

The differential diagnosis between osteoma and sesamoids, accessory or bipartite patella, inflammatory granuloma, loosened exostosis, calcified hematoma, and malignant tumor offers little difficulty. Joint chondromatosis frequently shows more or less extensive, though irregular, islands of calcification and rarely true bony elements. Chondromatous lesions are more frequently multiple than single, and they may occur anywhere within or without the joint capsule. Post-traumatic necrosis and sclerosis of Hoffa's fat pad never attain the size and density of an osteoma. The fat pad is either displaced or completely atrophied from long-standing pressure rather than the original site of a metaplastic bone formation (13, 14). The calcium content of the knee bones is usually normal, but increased functional impairment may lead to slight subchondral demineralization.

CONCLUSIONS

Benign osteoma of the knee joint is rare.

Roentgenograms show characteristic findings, differing from those in other arthrogenic tumors such as chondromatosis (joint mice). The absence of any bone connection between the tumor and the adjoining bony structures can readily be demonstrated. The history usually ranges over many years and often dates back to traumatism during the age of bone growth, with repeated mechanical damage to the knee in the interval. The size of the tumor as shown on roentgenograms and at operation is barely in proportion to the low-grade functional impairment and clinical complaints. Symptoms and signs seem to become more marked as the patient enters middle age.

The etiology of joint osteoma is still obscure. Our observations and those of

others (3, 12) seem to support the opinion of Kienböck that infection plays no part and that a single injury can hardly be the cause of the condition. Since repeated traumatism of occupational or athletic character can be traced in the majority of the patients, over many years, it can be assumed that mechanical influences induce a proliferation of a normally dormant, biologically polyvalent, and potentially bone-forming synovial tissue. Whether the primary damage of the synovial cells consists in a constitutional embryonal aberration or a traumatic detachment, with subsequent implantation before the termination of bone growth, cannot be decided. Insidious proliferation of dormant embryonal cell rests with metaplastic changes is well known in tumor pathology. A similar origin of the joint osteoma from synovial implantations under the impact of repeated mechanical damage over a long period of time conforms well with the present conception of tumor formation.

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Filing and Cross-Indexing of Radiation Therapy Records[1]

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IN A PREVIOUS publication (1) certain principles governing the filing and especially the cross-indexing of roentgen-ray diagnostic records were presented. These may be summarized as follows:

1. The clinical experience embodied in accumulated roentgen-ray records is of little value for either teaching or investigative purposes unless it can be approached in a systematic fashion, namely, through a sound method of cross-indexing.

2. A cross-indexing system is inextricably related to the method of handling the roentgen-ray records; indexing is valueless unless the records are filed so as to be readily available when located within the scheme of codification.

3. A cross-indexing system must be simple both in respect to the clerical effort involved and in the fineness of its subdivisions. Inordinately detailed categorization is impracticable and may result in the expenditure of excessive labor in maintaining the system. The primary objective of the cross-index is to simplify the tedious search through large masses of records for material pertinent to some particular purpose.

4. Division of all diagnoses to be classified into a number of broad diagnostic fields simplifies the coding scheme and minimizes labor. The field system automatically breaks down the mass of diagnoses into a number of groups, and the process of recovery of material is limited to a search within one of these rather than the total bulk of the records.

To translate these principles into practice, a coding scheme was presented which, with a minimum of clerical work, provided multiple diagnostic categories, within ten main divisions, of the abnormal findings possible in the field of diagnostic roentgenology. A separate code of normal findings, likewise divided into ten major fields, provided immediate segregation of normal from abnormal observations. The simplicity of the hand-scoring method and the ease of locating the desired diagnostic categories in the various fields (employing, in loose-leaf form, ruled normal and abnormal pages for each field) have made this method intensely practical and it has

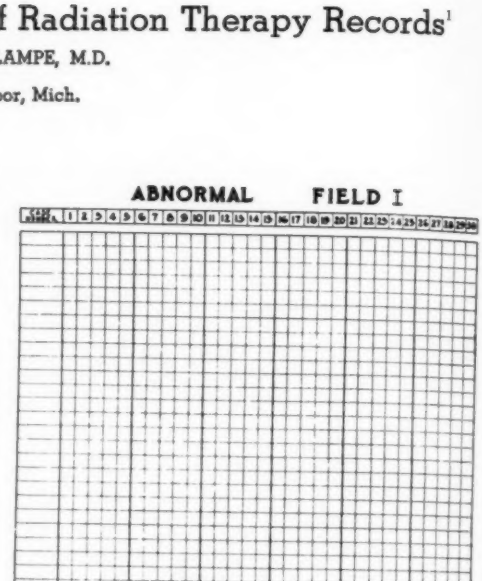

Fig. 1. Reproduction of the standard notebook page for indexing radiation therapy cases. This page is the same as that used for abnormal findings in the diagnostic cross-index. The form page has been numbered "I" for the Female Genital Tract.

[1] From the Department of Roentgenology, University Hospital, University of Michigan. Accepted for publication in February 1945.

gained considerable popularity among radiologists.

For the past four years in the Section on Radiation Therapy of the Department of Roentgenology at the University of Michigan, a cross-indexing system and coding plan based on principles identical with the diagnostic method and similar in external form have been employed with satisfactory results. With minor clerical effort the case records of all patients irradiated are catalogued into pertinent categories, distributed among a number of gross divisions (fields) of the entities encountered in radiation therapy practice. The mechanism of the system is such that, immediately upon cataloguing, the records are available for recall from the files for use in clinical investigation, teaching, or for other purposes. The indexed data recorded on form sheets (Fig. 1) by hand scoring lend themselves readily to tabulation of certain crude statistics on the number of cases treated. Since the case volume is usually considerably smaller in radiation therapy practice than in diagnostic roentgenology, in certain respects greater particularization is feasible and a means of providing this has been incorporated without altering the basic structure of the plan.

In Table I the major divisions of the code employed at the University of Michigan are listed. Perusal of this table shows that no attempt has been made to create an orderly classification of disease either on a histopathological or an anatomical basis. The divisions have been made largely but not exclusively on anatomical grounds; Fields X and XI and a portion of IX are the exceptions. The particular breakdown shown in this table is totally arbitrary and was made for reasons of convenience as dictated by local interests and past experiences. The number of fields, eleven, may appear unnecessarily high, but this was purposeful, in order to keep down the number of categories within each, permitting the introduction of the notation mechanism which is described later. These fields (and their subdivisions in Table II) are not presented as a scheme of universal applicability but rather to serve as an example of the application of the principles of the method. Each radiologist will undoubtedly find it to his advantage to establish a set of fields which more specifically meets the demands of his own practice.

TABLE I: MAJOR DIVISIONS OF THE RADIATION THERAPY CODE

FIELD I	Female Genital Tract
FIELD II	Male Genital Tract; Urinary Tract
FIELD III	Breast
FIELD IV	Lip and Mouth
FIELD V	Sinuses, Pharynx, Larynx, Thyroid
FIELD VI	Skin
FIELD VII	Gastro-intestinal Tract
FIELD VIII	Eye, Brain, Spinal Cord; Thorax
FIELD IX	Bone; Metastatic Neoplasm (Primary Unknown)
FIELD X	Lymphoblastoma; General
FIELD XI	Inflammations; Miscellany

In Table II the complete indexing code for radiation therapy cases is presented, listing the individual diagnoses used within each field along with their identifying sub-numbers. In order to distinguish clearly between field number and sub-number, the former is always written as a Roman numeral followed by an Arabic numeral as the sub-number; for example, "Carcinoma Cervix Uteri" is indicated by I-1. Study of this table shows that the diagnoses within the fields likewise do not follow any consistent histopathological or anatomical classification. The contents of the first ten fields are devoted predominantly to the various neoplasias, while Field XI is entirely constituted of non-neoplastic disorders, providing categories for a variety of inflammatory and miscellaneous conditions. This segregation is not absolute, since provision is made in the first ten fields for certain non-neoplastic entities. Examples of the latter are (1) "Disease Male Genital Tract XAL," Field II, category 6, which we have used for Peyronie's disease, sterilization for spermatocele, etc.; (2) Field V, category 13, "Disease Thyroid XAL," used for hyperthyroidism, thyroiditis, etc.

The diagnoses which appear within each field have been selected for a variety of reasons. Some have been chosen because of their high frequency of occurrence.

TABLE II: INDEXING CODE FOR RADIATION THERAPY CASES

Field I: Female Genital Tract

1. Carcinoma Cervix Uteri
2. Carcinoma Corpus Uteri
3. Neoplasm Uterus XAL
4. Carcinoma Vulva
5. Carcinoma Vagina
6. Neoplasm Ovary
7. Neoplasm Female Pelvis, Unspecified
8. Neoplasm Female Genital Tract XAL
9. Endometriosis (any site)
10. Castration, Uterine or Ovarian Disorder
11. Castration XAL
12. Disease Female Genital Tract XAL

Field II: Male Genital Tract

1. Carcinoma Penis
2. Carcinoma Scrotum
3. Carcinoma Prostate
4. Neoplasm Testicle
5. Neoplasm Male Genital Tract XAL
6. Disease Male Genital Tract XAL

Urinary Tract

10. Neoplasm Bladder
11. Neoplasm Kidney XAL
12. Wilms' Tumor
13. Neoplasm Urethra, Ureter
14. Neoplasm Urinary Tract XAL
15. Disease Urinary Tract XAL

Field III: Breast

1. Carcinoma Breast (no previous surgery), Preoperative Irradiation
2. Carcinoma Breast (no previous surgery), Inoperable (specify why)
3. Carcinoma Breast, Postoperative
   Specify type of operation: radical, modified radical, simple
4. Carcinoma Breast, Previous Treatment
   Recurrence or Metastases (specify involvement)
5. Neoplasm Breast XAL
6. Mastopathia Cystica
7. Disease Breast XAL

Field IV: Lip and Mouth

1. Carcinoma Lip, "massive concentrated" } Primary only treated
2. Carcinoma Lip, other irradiation } Primary only treated
3. Carcinoma Lip, irradiation of primary and metastases
4. Carcinoma Lip, XAL
5. Carcinoma Buccal Mucosa
6. Carcinoma Alveolar Mucosa
7. Carcinoma Palate
8. Carcinoma Floor of Mouth
9. Carcinoma Base of Tongue
10. Carcinoma Tongue XAL
11. Carcinoma Mouth, origin indeterminate
12. Neoplasm Lip, Mouth, Tongue XAL

Field V: Sinuses, Pharynx, Larynx, and Thyroid

1. Carcinoma Nose, except skin
2. Carcinoma Paranasal Sinuses
3. Neoplasm Nose and Sinuses XAL
4. Carcinoma Nasopharynx
5. Neoplasm Nasopharynx XAL
6. Neoplasm Auditory Canal, Middle Ear, Mastoid
7. Neoplasm Tonsil, Tonsillar Region
8. Carcinoma Pharynx, Hypopharynx
9. Carcinoma Larynx
10. Neoplasm Pharynx, Hypopharynx, Larynx XAL
11. Neoplasm Salivary Glands
12. Neoplasm Thyroid
13. Disease Thyroid XAL

Field VI: Skin

(Specify Histological Type)

1. Carcinoma Skin: Upper Extremity
2. Carcinoma Skin: Lower Extremity
3. Carcinoma Skin: Trunk
4. Carcinoma Skin: Neck
5. Carcinoma Skin: Nose
6. Carcinoma Skin: Ear
7. Carcinoma Skin: Scalp
8. Carcinoma Skin: Eyelids, Canthi
9. Carcinoma Skin: Face XAL
10. Hemangioma Skin, Subcutaneous Tissues
11. Neoplasm Skin XAL

Field VII: Gastro-Intestinal Tract

1. Neoplasm Esophagus
2. Neoplasm Stomach
3. Neoplasm Small Bowel
4. Neoplasm Colon, Rectum
5. Neoplasm Anus
6. Neoplasm Liver
7. Neoplasm Pancreas
8. Neoplasm Gastro-intestinal Tract XAL
9. Neoplasm Abdomen XAL
10. Neoplasm Retroperitoneum XAL (Adrenal)

Field VIII: Eye, Brain, Spinal Cord; Thorax

1. Carcinoma Conjunctiva
2. Carcinoma Orbit
3. Neoplasm Eye, Eyelid XAL
4. Disease Eye, Eyelid XAL
5. Pituitary Adenoma
6. Medulloblastoma
7. Neoplasm Brain XAL
8. Disease Brain XAL
9. Neoplasm Spinal Cord XAL
10. Disease Spinal Cord XAL

11. Carcinoma Bronchus
12. Neoplasm Lung XAL
13. Neoplasm Mediastinum XAL
14. Neoplasm Intrathoracic XAL
15. Neoplasm Thoracic Wall

Field IX: Bone

1. Giant-cell Tumor
2. Osteogenic Sarcoma
3. Chondrosarcoma
4. Ewing's Tumor
5. Sarcoma Bone XAL
6. Myeloma, Multiple Myeloma
7. Neoplasm Bone XAL
8. Xanthomatosis
9. Disease Bone XAL

Metastatic Neoplasm, Primary Unknown

10. Metastatic Neoplasm: Neck, Supraclavicular
11. Metastatic Neoplasm: Skull, Brain
12. Metastatic Neoplasm: Skeletal XAL
13. Metastatic Neoplasm: Lungs, Mediastinum, Hila
14. Metastatic Neoplasm: Abdomen
15. Metastatic Neoplasm: Retroperitoneal
16. Metastatic Neoplasm: Soft Tissues XAL

Field X: Lymphoblastoma; General

1. Hodgkin's Disease
2. Lymphosarcoma
3. Lymphoblastoma XAL
4. "Test of Irradiation" for Lymphoblastoma
5. Lymphatic Leukemia
6. Myelogenous Leukemia
7. Monocytic Leukemia
8. Leukemia XAL
9. Polycythemia
10. Disease Blood XAL
11. Hemangioma XAL*
12. Lymphangioma*
13. Melanoblastoma, Amelanotic Melanoblastoma*
14. Sarcoma XAL (organ site category preferable if available)
15. Neoplasm XAL

* Also use organ site category if available.

Field XI: Inflammations; Miscellany

1. Furuncle, Abscess, Carbuncle
2. Cellulitis, Phlegmon
3. Parotitis
4. Adenitis, Non-tuberculous
5. Mycotic and Unusual Infections: Actinomycosis, Blastomycosis, Rhinoscleroma, etc.
6. Adenitis, Tuberculous
7. Otitis Media, Peritubal Lymphoid Hypertrophy, Other Otological Infections
8. Other Inflammations or Infections
10. Thymic Enlargement
11. Keloid
12. Warts, Corns
13. Peptic Ulcer, Gastritis, Hyperacidity
14. Depression Salivary Secretion
15. Herpes Zoster, Neuralgias, etc.
16. Arthritis, Specify Type
17. Miscellany XAL

Others, which may occur relatively infrequently, appear because of a special interest on our part in these diseases. In other instances a relatively rare disease may be given a category to itself to avoid searching through a large number of records of a frequently occurring closely related disorder. Those familiar with the technic of setting up an inclusive coding mechanism will recognize the use of certain categories for the sole purpose of providing pigeonholes which will sweep up situations not provided for elsewhere but which do not merit individual consideration. As stated previously, this indexing code is not presented with the thought that it will prove to be equally satisfactory for all radiation therapy practices, but rather as an example of the application of this method. Radiologists who include the radiation treatment of dermatitides and dermatoses in their work will find no provision for these diseases since at our institution this work is carried on in another department. These diseases can be added readily by means of an additional field or by incorporating the necessary categories into Field VI, which deals with skin.

In order to insure efficient operation of this coding scheme, several simple principles must be faithfully observed. Of paramount importance is the fact that the plan calls for handling of the neoplastic entities on the basis of the primary site of the lesion. This principle holds regardless of whether the primary lesion or its metastases or both have been treated; in any of these events, the case is indexed in the category bearing the label of the primary tumor. For those cases presenting obvious metastases derived from an unknown primary tumor, provision for codification is found in Field IX, categories 10 to 16, inclusive; these diagnoses are not used to specify the sites of metastasis from a known primary lesion.

Throughout the code, the terms "carcinoma" and "neoplasm" are used in their precise senses and are not interchangeable. A malignant neoplasm which is not a carcinoma must not be placed in a category labeled "carcinoma" of the involved part but in a suitable category bearing the specific histological diagnosis or in one labeled "neoplasm." Neoplasm, of course, includes all types of neoplasia. For example, a sarcoma of the body of the uterus is catalogued as Field I-3 ("Neoplasm Uterus XAL"); it is not entered in I-2 ("Carcinoma Corpus Uteri"). Any neoplasm of the testicle: sarcomatous, carcinomatous, mixed, benign or malignant, is coded as II-4 ("Neoplasm Testicle"). The Wilms' tumor has its own category, II-12, while renal hypernephromas and carcinomas are cared for with II-11 ("Neoplasm Kidney XAL").

The designation "XAL" as it appears in Table II may be translated "except as

listed elsewhere in the code." "Lymphoblastoma XAL" (X-3) refers to any type of lymphoblastoma other than those listed, namely, "Hodgkin's disease" (X-1), "lymphosarcoma" (X-2), and the leukemias (X-5, 6, 7, 8). "Neoplasm Mediastinum XAL" (VIII-13) provides for mediastinal tumors other than lymphoblastomatous masses, since the latter are codified in Field X. A final example of the use of the "XAL" designation is X-15 entitled "Neoplasm XAL"; this is a pigeonhole purposefully created to house an occasional situation not provided for elsewhere in the code and in our experience has been used rarely.

Although the standard form[2] employed (Fig. 1) provides space for as many as thirty categories within each field, the radiation therapy code within each of its fields never calls for this maximal number. Field XI, containing seventeen categories, requires seventeen of the thirty columns; the other fields use less. This leaves space on the right-hand side of each page which is employed to advantage by entering brief notes. These notations can serve to provide greater specificity than is inherent in the category title or may be used to call attention to striking or interesting features of any case. In Fields III, VI, and XI, the code calls for specification by means of notes of such items as the reason for inoperability in the case of mammary tumors, the type of operation, the sites of recurrence and metastasis, and the histological type of cutaneous carcinomas. The notation feature may be used to specify the precise histologic character of a lesion whenever the category bears the broad label of neoplasm and also to specify location when this does not appear in the category title. Examples of the former are: I-6, adenocarcinoma (the title of this category is "Neoplasm Ovary," the notation "adenocarcinoma" gives the precise histological type); V-7, carcinoma (the label of this category is "Neoplasm Tonsil"). Examples of specification of anatomical location are: VI-10, cheek (this category is labeled "Hemangioma Skin, Subcutaneous Tissues"; the notation "cheek" states the precise site); IX-4, femur (thus locating the Ewing's tumor in the femur); XI-1, face (indicating the presence of a furuncle on the face).

Obviously the notation scheme may be used to indicate the type of treatment or the chronological relation of irradiation to operation. Spectacular or unexpectedly poor responses to irradiation may be labeled; unusual age incidence and many other items of interest may be incorporated to facilitate subsequent location of such material. The notation feature is elastic and may be employed as broadly or as narrowly as one's interests dictate.

In practice it has been our custom to codify each case at the time of completion rather than at the beginning of a treatment program. On the patient's radiation therapy record, near the discharge note, the coded statement appears, Roman numerals being employed for field designation and Arabic numbers for the diagnosis within the field. The entering of this material on the standard forms is usually done daily. A loose-leaf notebook containing eleven standard forms, one for each field, properly labeled as to field number, is maintained. The case number is entered in the left-hand column; in the proper column an X is hand-scored and on the right side of the page the desired notations are inscribed. With the 1,300 to 1,500 patients treated each year in our department, it has been found that five to ten minutes daily are required to code and record the cases; a negligible investment of time which yields rich rewards in ability to recover valuable clinical material.

To illustrate the technic and appearance of the completed coding and recording process in the various field forms, Figure 2 and Table III are presented. In Figure 2 samples of indexed cases from various fields are shown: the case number identi-

[2] This form is the same as that used for abnormal findings in the indexing scheme for diagnostic roentgenology and may be obtained from Edwards Bros., Inc., Ann Arbor, Mich. It was more convenient to use this existing form than to create a special form for the radiation therapy code.

fying the patient appears in the left-hand column, the hand-scored X is present in the column corresponding to the number of the diagnosis as found in the indexing code (Table II), and on the right side of the sheet are various notations. Table III, dealing with the cases shown in Figure 2, may serve to translate and amplify the material recorded in this figure. The diagnosis is compared with the category label of the indexing code (Table II) and the coded statement for each diagnosis is given along with the notations used in these cases.

One feature regarding case records in radiation therapy must be noted. With an indexing system such as is presented here, it is eminently desirable that each case be characterized by a single case number which is reassigned to the patient at whatever future date he may return. The particular form of the radiation therapy record may vary widely. The record at the University Hospital consists of 3 elements: (1) the original refer if the case is referred from another department for radiation treatment; (2) the treatment sheets containing the detailed data of physical factors, dosage, and location of sites for roentgen and radium therapy; (3) the sheets devoted to history, physical findings, radiation prescriptions, clinical progress notes, and discharge notes. The three elements are kept in a single envelope bearing the patient's name and registration number and are maintained on file in the radiation therapy department. All the material in the record is created in duplicate as the record develops, and the copies appear in the general hospital record of the case. Thus the complete radiation therapy story of any case, including detailed physical factors of treatment, is available in the patient's hospital record as well as in the record housed in the Department of Roentgenology. We believe that this must be done in fairness to the patient, the hospital, and all the physicians involved.

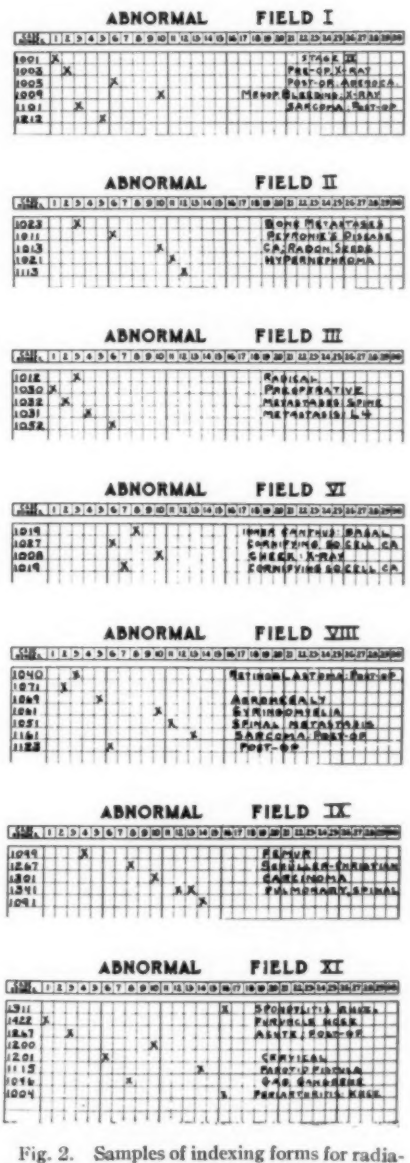

Fig. 2. Samples of indexing forms for radiation therapy cases for various fields. Coding examples in Table III have been entered here to demonstrate the completed process of cross-indexing.

As in the system previously described for diagnostic roentgenology, total incidence figures are readily obtainable for any desired finite period by a simple count of the scores within any pertinent column. It has been found convenient to separate the months by entering the month date to the left of the case number column

TABLE III: SAMPLE OF CODING AND RECORDING

Case No.	Diagnosis	Category Label	Code	Notation
		Field I		
1001	Carcinoma cervix uteri	Same	I-1	Stage IV
1003	Carcinoma corpus uteri	Same	I-2	Preoperative roentgen irradiation
1005	Adenocarcinoma of ovary	Neoplasm Ovary	I-6	Adenocarcinoma, postoperative
1009	Menopausal bleeding	Castration (Uterine or Ovarian Disorder)	I-10	Menopausal bleeding, roentgen irradiation
1101	Sarcoma of uterus	Neoplasm Uterus XAL	I-3	Sarcoma, postoperative
1212	Carcinoma of vagina	Same	I-5	
		Field II		
1023	Carcinoma of prostate	Same	II-3	Bone metastases
1011	Peyronie's disease	Disease Male Genital Tract XAL	II-6	Peyronie's disease
1013	Carcinoma of bladder	Neoplasm Bladder	II-10	Carcinoma; radon seeds
1021	Hypernephroma of kidney	Neoplasm Kidney XAL	II-11	Hypernephroma
1113	Wilms' tumor	Same	II-12	
		Field III		
1012	Carcinoma of breast, postoperative	See Table II, Field III-3	III-3	Radical operation
1030	Carcinoma of breast, preoperative irradiation	See Table II, Field III-1	III-1	Preoperative
1032	Carcinoma of breast, spinal metastases	See Table II, Field III-2	III-2	Metastases spine
1031	Carcinoma of breast, spinal metastasis (previous surgery)	See Table II, Field III-4	III-4	Metastasis L4
1052	Mastopathia cystica	Same	III-6	
		Field VI		
1019	Basal-cell carcinoma of inner canthus	Carcinoma Skin: Eyelids, Canthi	VI-8	Inner canthus, basal
1027	Cornifying squamous-cell carcinoma of skin of ear	Carcinoma Skin: Ear	VI-6	Cornifying squamous-cell carcinoma
1008	Cavernous hemangioma, cheek	Hemangioma Skin, Subcutaneous Tissues	VI-10	Cheek; x-ray
1019	Cornifying squamous-cell carcinoma of scalp	Carcinoma Skin: Scalp	VI-7	Cornifying squamous-cell carcinoma
		Field VIII		
1040	Retinoblastoma right eye, postoperative	Neoplasm Eye, Eyelid XAL	VIII-3	Retinoblastoma, postoperative
1071	Carcinoma of orbit	Same	VIII-2	
1069	Acromegaly	Pituitary Adenoma	VIII-5	Acromegaly
1061	Syringomyelia	Disease Spinal Cord XAL	VIII-10	Syringomyelia
1051	Carcinoma of bronchus with spinal metastasis	Carcinoma Bronchus	VIII-11	Spinal metastasis
1161	Sarcoma of mediastinum, postoperative	Neoplasm Mediastinum	VIII-13	Sarcoma, postoperative
1123	Medulloblastoma, postoperative	Medulloblastoma	VIII-6	Postoperative
		Field IX		
1099	Ewing's tumor of femur	Ewing's Tumor	IX-4	Femur
1267	Schüller-Christian's disease	Xanthomatosis	IX-8	Schüller-Christian
1301	Metastatic carcinoma in neck, primary unknown	Metastatic Neoplasm: Neck, Supraclavicular	IX-10	Carcinoma
1341	Pulmonary and spinal metastases, primary unknown	Metastatic Neoplasm: Skeletal XAL Metastatic Neoplasm: Lungs, Mediastinum, Hila	IX-12, 13	Pulmonary, spinal
1091	Abdominal metastases, primary unknown	Metastatic Neoplasm: Abdomen	IX-14	

Field XI

1311	Spondylitis rhizomélique	Arthritis	XI-16	Spondylitis rhizomélique
1422	Furuncle, nose	Furuncle, Abscess, Carbuncle	XI-1	Furuncle, nose
1267	Acute postoperative parotitis	Parotitis	XI-3	Acute, postoperative
1200	Thymic enlargement	Same	XI-10	
1201	Tuberculous adenitis, cervical	Adenitis, Tuberculous	XI-6	Cervical
1115	Parotid fistula	Depression Salivary Secretion	XI-14	Parotid fistula
1046	Gas gangrene	Other Inflammations or Infections	XI-8	Gas gangrene
1004	Periarthritis of knee	Arthritis	XI-16	Periarthritis, knee

at the beginning of each new month. For statistical purposes it has been desirable to indicate repeated episodes of irradiation within the calendar year by means of an asterisk immediately to the left of the case number; to obtain counts of the number of patients, those numbers marked with an asterisk are omitted. Likewise, one may indicate by means of another symbol cases which were treated in previous years. It is possible at the end of a calendar year to obtain a statistical survey of the number of patients treated for the various diseases catalogued, by the expenditure of only a few hours in tabulating column counts. This procedure is carried out yearly in our department; usually by the end of the first week in January the survey of the entire preceding year is prepared and may be compared with the experience of the earlier years.

The recovery of case material from the hand-scored sheets is a matter of utter simplicity. The radiologist turns to the proper field sheet in the loose-leaf book, locates the column bearing the desired diagnostic number, and scans it for the hand-scored X's. The corresponding case number is found in the left-hand column and the case record may then be located in the files on the basis of this number. This may be achieved by maintaining the records in the file in serial order of case number. If it is deemed advisable to file the records in alphabetical order by name, a number-name card index will provide for transition from case number to patient's name.

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REFERENCE

1. HODGES, FRED J., AND LAMPE, ISADORE: Filing and Cross-Indexing Roentgen Ray Records. Am. J. Roentgenol. **41**: 1007–1018, June 1939.

Cross-Indexing of Roentgen Diagnostic Records[1]

A Revision of the Hodges and Lampe Code

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WHEN THE roentgenologist has exposed and developed his second roentgenogram the task of keeping track of his accumulated experience begins. At that stage the problem is a simple one. The location of the two films with their findings can easily be kept in mind and either can be located for comparison with future examinations. The sum total of that individual's recorded roentgenological experience is immediately available.

As examination follows examination, the situation becomes vastly different. The present-day volume of roentgenological work is so large that each day's cases are soon submerged by the continuing flow. The interesting cases are lost sight of, and the surprisingly large experience represented by thousands of examinations is not available for orderly review. To prevent this loss, a systematic means of access to the accumulated material becomes a necessity in every roentgenological office and department.

Various systems of cross-indexing have been applied to this problem. The most common is the card-index system, which has many variations, all based on the same principle. A card is prepared for each diagnosis, and the diagnosis is indicated at the top by title or by a code number. If the method is to be practicable, the less common diagnoses must be grouped several to a card in order to decrease the number of cards and thus simplify the clerical work. Every day the names or file numbers of the patients are written in on the proper cards.

The card-index method is direct. Its chief disadvantage is that a separate card must be maintained for each diagnostic category. If all the possible diagnoses are grouped into 300 categories, for instance, 300 cards must be used, and the clerical work of entering cases on the cards is correspondingly complex.

In 1939 Hodges and Lampe (1) introduced the loose-leaf "score sheet" principle as a means of simplification. They replaced the library card by a score sheet 8 1/2 by 11 inches, ruled into 30 vertical columns and 50 horizontal columns.[2] The horizontal column is thus divided into 30 squares. Each vertical column represents a diagnosis or diagnostic category and is identified by a number. Each horizontal column represents one examination and is identified by writing the patient's file number in the left-hand space. The diagnosis made on that case is then indicated by a check mark or other symbol placed in the square at the intersection of the horizontal column with the proper vertical column. The score sheet, in comparison to the index card, may be said to be two-dimensional, since it will accommodate many diagnoses as well as many patients. Thirty diagnostic categories are included on each sheet, making it possible to use only 10 sheets for 300 diagnostic categories.

To use these score sheets Hodges and Lampe divide all roentgenological diagnoses into 10 major anatomic groups, which they term "fields," and further subdivide each field into 30 categories. The more common diagnoses are represented by separate categories; the less common diagnoses are grouped several to a category. The entire gamut of roentgenological diagnoses is thus divided into 300 categories, grouped in 10 anatomic fields and tabulated on 10 sheets.

A second parallel system of eight anatomic fields and eight more score sheets is

[1] From the Radiological Service, O'Reilly General Hospital, Springfield, Mo. Accepted for publication in December 1944.

[2] Available in printed form from Edwards Bros., Ann Arbor, Mich.

also provided, with which to index negative findings, classified according to the type of examination.

At the close of the dictation of each report, the radiologist refers to the code and dictates a code number which is typed on the report and serves as a guide to the stenographer who later enters the case on the appropriate score sheet. This code number consists of a Roman numeral to indicate the field and an Arabic numeral to indicate the diagnostic category within the field. The Roman numerals therefore run from I through X and the Arabic numerals from 1 through 30. An asterisk replaces the check mark in the appropriate space to emphasize a case of unusual interest, while a question mark qualifies an uncertain diagnosis. Alphabetical letters are provided in some fields to further specify location. When needed, they are dictated following the code number. The proper column on the score sheet is then marked by writing in the letter instead of by the usual check mark.

Since its introduction, the score-sheet cross-indexing system has been adopted in many roentgenological offices and departments. It has proved capable of handling a large volume of material without an undue clerical burden. But, as Hodges and Lampe pointed out, the cross-index must be tailored to fit the type of work being done and the particular use to which it is to be put. Thus it is to be expected that those using the cross-index will arrive at individual variations, both in usage and in construction of the code. It is to be emphasized that each radiologist, after experience with the code system, can profitably make his own personal variations. Periodic revision also is necessary to bring the code up to date and to adapt it more efficiently to the work being done. Such a revision is presented here, together with a discussion of how the code may be used for various purposes.

SUGGESTED CHANGES IN CODE

1. The separation of the diagnoses into 10 anatomic fields provides, in effect, 10 codes of 30 numbers each. Many diagnoses will occur in more than one of these fields. If the same code number is assigned in the various fields for those diagnoses which occur in more than one field, it will speed up the coding process by making it easier to remember the numbers.

Although this concept is followed to a certain extent in the Hodges and Lampe code, there are numerous violations. For example, they code a recent fracture as 12 or 18 in Field I, 10 in Field III, and 2 in Fields II, IV, and V. In the code published herewith, fracture is coded as number 3 in no matter what field it occurs—bones of face, skull, spine, or extremities. The single relationship "fracture is 3" is more easily remembered than "fracture is 12, 18, 10, or 2." In the revised code, identical access numbers have been provided for similar diagnoses in all fields throughout the code.

2. The second change in the code is the alteration of certain categories to keep up with recent progress in medical concepts. This reclassification was carried farthest in Field VII (Lungs and Pleura, non-tuberculous). The previous classification of non-tuberculous pulmonary infections included:

6. Pneumonia, lobar
7. Pneumonia, resolving or resolved
8. Pneumonia, unresolved
9. Pneumonia, except as above
14. Pneumonitis, suppurative
15. Pneumonitis, except as above
16. Abscess, cyst

The revised classification of this group is as follows:

3. Bronchopneumonia and primary atypical pneumonia
4. Lobar pneumonia
5. Abscess and suppurative pneumonitis
6. Pneumonia and pneumonitis, except as above (including fungus, sarcoid, tularemic and chronic fibroid pneumonia)

It is felt that there will be less variation in coding cases under this latter classification and that, therefore, the code will be of more value when it is eventually called upon to furnish cases for review or demonstration.

Other minor shifts in wording and grouping of the diagnostic categories have been made throughout the code. For the convenience of Army radiologists a code number for foreign body has been provided in every field except that of Thoracic Tuberculosis.

3. A mechanism has been added to separate the "repeat" diagnoses of follow-up examinations from the "new" diagnoses. This is of great practical importance to avoid reduplication in preparing lists of cases from the score sheets. In the Hodges and Lampe code, duodenal ulcer is indicated by IX-7. To prepare a list of cases of duodenal ulcer for review, one includes all the cases checked in column 7 on the Field IX sheets. One will find, however, that many of the examinations listed as IX-7 are follow-up examinations and, as a result, the same cases will recur several times in the list prepared. When finding groups of cases for review, these reduplications are readily recognized and are simply an annoyance. They are a decided handicap, however, in preparing statistics since, instead of merely counting the check marks in a given vertical column, it is necessary to make note of all the case numbers and to weed out carefully all the reduplications.

In the revised code presented herewith the follow-up examinations are differentiated from the new cases by the addition of the letter X to the code number of any diagnosis which has been previously coded on the same patient. These diagnoses are entered on the score sheet by a cross instead of a check mark. Thus the radiologist can determine how many duodenal ulcers he has diagnosed in the past year, for example, by simply counting the check marks in the proper column, omitting the crosses.

4. Question marks and asterisks are used in the original code to qualify questionable diagnoses and emphasize cases of special interest. This sometimes makes it necessary to crowd several symbols into one box. In the revised code this is avoided by assigning these functions to special vertical columns (numbers 28 and 30).

5. The Hodges and Lampe system codes all examinations with normal findings according to the type of examination performed, but it does not provide this information for the abnormal cases. Frequently the type of examination is obvious from the diagnosis, but in other cases this is not so. For instance, a gallbladder calculus may be found on an otherwise negative intravenous pyelogram. Such a case is coded as gallbladder calculus and will not be listed anywhere in the code as a pyelographic study. The only purpose of coding negative examinations is statistical, and it is of no value unless the comparable figures (classified in the same way, by examination type) are available for the abnormal cases.

In our revised code, therefore, a provision is made (by letter in column 2) for indicating the type of examination in the abnormal cases. Thus, an example of the full code indication is: VI-2 E, 13. On sheet VI an E will be entered in column 2 to indicate a portable chest examination (and column 13 will be checked to indicate cardiac enlargement). In this way a record of the examination type can be made for all cases.

6. The second system of eight score sheets used by Hodges and Lampe for recording the type of examination in negative cases has been eliminated by using column 1 in each field for this purpose just as column 2 is used to code the examination type on abnormal examinations. The number of score sheets has thus been reduced from 18 to 10.

SUGGESTED VARIATIONS IN USE OF THE CODE

Most radiologists who use the score-sheet method for cross-indexing their reports are coding all of their examinations. For many of them, depending on the use to be made of the code, this is unnecessary and time-wasting. The actual use made of the cross-index has fallen into three common forms, depending on the interest and available study time of the individual radiologist:

A. The index may be used merely to locate unusually interesting and instructive cases for demonstration and teaching, or for comparison with similar cases seen later. Used in this way, the cross-index fills the purpose of the "Special File" in which nearly every radiologist keeps his most cherished cases. In many war-time offices this is the only function of the cross-index utilized; yet even this limited use of the code may be a source of great satisfaction to the radiologist.

If this is the only use anticipated, it is obvious that only cases of particular interest need be coded and entered on the score sheets. Even in these cases the examination type may be omitted and only the diagnosis coded. This simplified usage has the advantage of eliminating 98 per cent of the clerical work involved in full use of the code.

B. The index may be used to locate series of cases for review and study. This purpose requires a more complete utilization of the code than does use A. It will be necessary to code *all* of the abnormal examinations. Negative examinations, however, need not be coded, the repeat examinations may be omitted, and only the diagnoses are needed (not the examination types). Columns 1 and 2 may, therefore, be omitted. The clerical work will be approximately 30 per cent of that involved in full usage of the code.

C. The fullest use of the cross-index is for periodic statistical study. This necessitates coding every case examined, using the examination type columns, 1 and 2, as well as the diagnosis columns. Simple counting on the score sheets for any given period will then determine the total number of examinations or of patients (omitting re-examinations), the number of examinations (or patients) of each type, the relation of negative to positive results for each type of examination, the number of times any diagnosis has been made, the relative frequency of various diagnoses, etc. Followed from year to year, the score sheet will also indicate the trend in volume of the various types of work being done. In the case of a hospital department, this information may be particularly helpful in supporting requests for changes and additions in departmental equipment and personnel.

Each roentgenologist using the code must determine for what purposes he will employ it and should use only those portions which are necessary for his object.

TECHNIC OF USING THE CODE

At the close of dictation of each x-ray report, the code numbers of the case are dictated by the radiologist. The stenographer types these in a lower corner of the report and later transcribes all of the day's codes to the score sheets. The field numbers and diagnosis numbers are self-explanatory and remain the same regardless of how the code is being used.

Each field contains a listing of letters headed "Examination Type or Localization." In some fields separate lists are given for these two purposes. The use of these letters will vary with the different methods of using the code. If normal cases are to be coded they are always listed as "1" in the proper field, followed by the proper letter to indicate the type of examination performed. (Example: Negative chest stereo. Dictated: "Six dash 1A." Written on report: VI-1A. Entered on score sheet by placing the patient's file number in the left-hand space of a horizontal column of the field VI sheet and placing A in vertical column 1.)

The coding of the examination type in an abnormal case is exactly similar, but uses column 2 instead of column 1 and is followed by the diagnosis code number. (Example: Fracture of the femur. Dictated: "Five dash 2C comma 3." Written on report: V-2C, 3. Entered on score sheet by placing patient's file number in the left-hand space of a horizontal column, C in vertical column 2, and a check mark in vertical column 3.) If the case is a follow-up examination and the fracture has previously been coded, the diagnosis number is followed by X. (The previous example would become: "Five dash 2C

comma 3X." V-2C, 3X. It would be scored as above, but an X would replace the check in column 3.)

Occasionally, even after the examination type has been coded, there is need for further localization of the disease process. This is accomplished by using the localizing letter in the diagnosis column. (Example: Ureteral calculus demonstrated by excretory pyelogram. Dictated: "Ten dash 2B comma 19C." Written: X-2B, 19C. Entered on score sheets by placing B in column 2 and C in column 19.)

If the examination types are omitted, the localization must be used more often. In the case of the femoral fracture previously cited, it would be necessary to use the localizing letter C following the diagnosis number. ("Five dash 3C." V-3C. Column 2 on the score sheet is left blank and C is entered in column 3.)

It is not necessary to use a localizing letter following the diagnosis number if the site of the lesion is explained either by the examination type or by the wording of the diagnostic category. (Example: Field I-7 refers specifically to clouding of the sinuses and no localizing letter is necessary even though the examination type is not being coded.)

It is suggested that fractures of epiphyseal portions of the long bones be localized by the letter of the joint near which they occur instead of by the bone involved. This will separate them from fractures of the shaft.

Questionable diagnoses and cases of special interest are emphasized by coding number 28 or 30 in addition to the diagnosis number.

The diagnostic category number 27, "except as above," occurs in each field. This is a wastebasket category to include all the rare diagnoses not specifically listed. When "except as above" is used, it should be followed by a simple statement of the diagnosis to be written on the score sheet. (Example: Esophageal varices. Dictated: "Nine dash 27 esophageal varices." Written IX–27 esophageal varices. A check mark is entered in column 27 of Field IX and the words "esophageal varices" are written in the same horizontal column.) This procedure makes it possible to locate rare cases quickly in the score sheets without having to devote an entire diagnostic category to each one. Since such diagnoses are infrequent, they do not materially increase the clerical work in coding.

REVISED CODE FOR DIAGNOSTIC RECORDS

Field I: Bones of Face, Orbit, Mandible, Temporomandibular Joints, Sinuses, Mastoids, Teeth

1. Negative (Code examination type)
2. Abnormal (Code examination type. Do not code 2 if examination type is to be coded in another field.)

Bones of face, orbit, and mandible

3. Fracture
4. Deviation of nasal septum
6. Osteomyelitis

Sinuses

7. Clouding
8. Thickening of mucous membrane
9. Polyp, mucocele

Mastoids

11. Clouding
12. Thickening of cell walls
13. Sclerosis
14. Bone destruction
15. Cholesteatoma

Teeth

16. Impaction or other abnormal position
17. Caries
18. Periapical abnormality

20. Foreign body, soft tissue abnormality
21. Benign neoplasm
22. Malignant neoplasm, primary
23. Malignant neoplasm, secondary
24. Anomaly
25. Postoperative
26. Sinus tract
27. Except as above
28. Diagnosis questionable (Code probable diagnosis also)
29. Unsatisfactory examination
30. Special interest

Examination type or localization (use localization only when necessary)

A. Skull
B. Sinuses
C. Mastoids

D. Bones of face
E. Mandible
F. Temporomandibular joints
G. Orbit
H. Teeth

P. Except as above

Follow the diagnosis number by X if the diagnosis has been coded on a previous examination.

Field II: Skull

1. Negative (Code examination type)
2. Abnormal (Code examination type. Do not code 2 if examination type is to be coded in another field.)
3. Fracture
4. Physiological calcification
5. Benign hyperostosis
6. Inflammatory disease of bone
7. Paget's disease, osteitis fibrosa cystica, hyperparathyroidism, osteopetrosis

Evidence of intracranial lesion

8. Sutural changes, accentuated convolutional markings, hydrocephalus
9. Intrasellar erosion
10. Extrasellar or parasellar erosion
11. Local bone changes, destructive or proliferative
12. Displacement of physiological calcification
13. Abnormal intracranial calcification
14. Unusual vascular markings suggesting intracranial lesion
17. Intracranial tumor, supratentorial
18. Intracranial tumor, infratentorial
19. Intracranial tumor, unspecified
20. Foreign body, soft-tissue abnormality
21. Benign neoplasm
22. Malignant neoplasm, primary
23. Malignant neoplasm, secondary
24. Anomaly
25. Postoperative
26. Sinus tract
27. Except as above
28. Diagnosis questionable (Code probable diagnosis also)
29. Unsatisfactory examination
30. Special interest

Examination type or localization

A. Skull
B. Encephalogram
C. Ventriculogram

P. Except as above

Follow the diagnosis number by X if the diagnosis has been coded on a previous examination.

Field III: Spine

1. Negative (Code examination type)
2. Abnormal (Code examination type. Do not code 2 if examination type is to be coded in another field.)
3. Fracture
4. Abnormality of the intervertebral disk
6. Osteomyelitis
7. Paget's disease, osteitis fibrosa cystica, hyperparathyroidism, osteoporosis causing disability
9. Spondylolisthesis
10. Dislocation, subluxation
11. Epiphyseal separation
12. Osteochondritis (epiphysitis)
13. Spondylitis rhizomélique
14. Hypertrophic spondylitis
15. Trophic arthritis (Charcot joint, syringomyelia)
16. Arthritis, mechanical basis
17. Pott's disease
18. Abnormal alignment of the spine
20. Foreign body, soft tissue abnormality
21. Benign neoplasm
22. Malignant neoplasm, primary
23. Malignant neoplasm, secondary
24. Anomaly
25. Postoperative
26. Sinus tract
27. Except as above
28. Diagnosis questionable (Code probable diagnosis also)
29. Unsatisfactory examination
30. Special interest

Examination type or localization (*Use localization only when necessary*)

A. Cervical spine
B. Dorsal spine
C. Lumbar spine and sacrum
D. Dorsal and lumbar spine
F. Myelogram

P. Except as above

Follow the diagnosis number by X if the diagnosis has been coded on a previous examination.

Field IV: Upper Extremity. Field V: Lower Extremity

1. Negative (Code examination type)
2. Abnormal (Code examination type. Do not code 2 if examination type is to be coded in another field.)
3. Fracture, recent
4. Fracture, healing or healed
5. Fracture, non-union or malunion
6. Osteomyelitis
7. Paget's disease, osteitis fibrosa cystica, hyperparathyroidism, osteopetrosis, osteoporosis causing disability, osteogenesis imperfecta, metabolic bone change

8. Rickets, scurvy, lead line, congenital lues, achondroplasia
9. Ankylosis
10. Dislocation, subluxation
11. Slipped epiphysis, epiphyseal separation
12. Osteochondritis, osteochondritis dissecans, loose body
13. Arthritis, atrophic
14. Arthritis, hypertrophic
15. Arthritis, trophic, Charcot, syringomyelia
16. Arthritis, mechanical basis
17. Tuberculosis, bone or joint
18. Arthritis, purulent, septic, gonococcal
19. Arthritis, metabolic, gout, hemophilia, etc.
20. Foreign body, soft-tissue lesion
21. Benign neoplasm
22. Malignant neoplasm, primary
23. Malignant neoplasm, secondary
24. Anomaly
25. Postoperative
26. Sinus tract
27. Except as above
28. Diagnosis questionable (Code probable diagnosis also)
29. Unsatisfactory examination
30. Special interest

Examination type or localization. (Localize fractures of the diaphysis by the bone letter; of the metaphysis by the joint letter.)

Upper Extremity

A. Clavicle and sternoclavicular joint
B. Shoulder joint and scapula
C. Humerus
D. Elbow
E. Radius and ulna
F. Wrist
G. Hand
P. Except as above

Lower Extremity

A. Pelvis and sacro-iliac joint
B. Hip
C. Femur
D. Knee
E. Tibia and fibula
F. Ankle
G. Foot
P. Except as above

Follow the diagnosis number by X if the diagnosis has been coded on a previous examination.

Field VI: Thoracic Cage, Mediastinum, Soft Tissues of Neck, Cardiovascular System

1. Negative (Code examination type)
2. Abnormal (Code examination type. Do not code 2 if examination type is to be coded in another field.)

Thoracic cage

3. Fracture
4. Inflammatory disease of bone

Mediastinum

5. Non-specific inflammatory lymphadenopathy
6. Abnormal thymus
7. Abnormal thyroid
8. Non-neoplastic abnormality of larynx
9. Mediastinitis, retropharyngeal adenitis or abscess
10. Lymphoblastoma (Code also under 22 or 23)

Cardiovascular system

12. Pulmonary hyperemia, edema
13. Generalized cardiac enlargement
14. Characteristic contour of heart, abnormal pulsation
15. Pericardial effusion
16. Chronic adhesive pericarditis, calcification of pericardium
17. Calcification, myocardium or heart valves
18. Aneurysm, luetic aortitis
19. Tortuous, elongated aorta, calcification of aorta
20. Foreign body, soft tissue abnormality
21. Benign neoplasm
22. Malignant neoplasm, primary
23. Malignant neoplasm, secondary
24. Anomaly, including congenital heart disease
25. Postoperative
26. Sinus tract
27. Except as above
28. Diagnosis questionable (Code probable diagnosis also)
29. Unsatisfactory examination
30. Special interest

Examination type

A. Chest, stereo
B. Chest, single film
C. Chest, grid film
D. Chest, lateral or oblique
E. Chest, portable
F. Chest, thymus technic
H. Heart and aorta
I. Soft tissues of neck
J. Chest fluoroscopy
P. Except as above

Localization (use only when necessary)

A. Thoracic cage
B. Mediastinum
H. Heart and aorta
I. Soft tissues of neck
P. Except as above

Follow the diagnosis number by X if the diagnosis has been coded on a previous examination.

Field VII: Lungs and Pleura (Non-Tuberculous)

1. Negative (Code examination type)
2. Abnormal (Code examination type. Do not code 2 if examination type is to be coded in another field.)
3. Bronchopneumonia, primary atypical pneumonia
4. Lobar pneumonia
5. Abscess, suppurative pneumonitis
6. Pneumonia and pneumonitis, except as above (include fungus, sarcoid, tularemic, chronic fibroid pneumonia, etc.)
10. Bronchiectasis
11. Abnormal bronchogram other than bronchiectasis
12. Pulmonary emphysema, pneumonocele
13. Pneumoconiosis
14. Radiation fibrosis
15. Atelectasis
16. Pneumothorax, induced, spontaneous, traumatic
17. Tuberculosis, differential diagnosis only (Code 28 also)
18. Pleuritis without effusion
19. Pleural effusion, empyema
20. Foreign body, soft tissue abnormality
21. Benign neoplasm
22. Malignant neoplasm, primary
23. Malignant neoplasm, secondary
24. Anomaly
25. Postoperative
26. Sinus tract
27. Except as above
28. Diagnosis questionable. (Code probable diagnosis also)
29. Unsatisfactory examination
30. Special interest

Examination type

A. Chest, stereo
B. Chest, single film
C. Chest, grid film
D. Chest, lateral or oblique
E. Chest, portable
F. Chest, thymus technic
G. Bronchogram
I. Soft tissues of neck
J. Chest fluoroscopy

P. Except as above

Follow the diagnosis number by X if the diagnosis has been coded on a previous examination.

Field VIII: Intrathoracic Tuberculosis

2. Abnormal (Code examination type. Do not code 2 if examination type is to be coded in another field.)
3. Calcified primary lesion, parenchymal
4. Calcified primary lesion, lymph node
5. Non-calcified primary lesion, parenchymal or lymph node
6. Tuberculosis suspect, existence of lesion questionable
7. Minimal pulmonary tuberculosis
8. Moderately advanced pulmonary tuberculosis
9. Far advanced pulmonary tuberculosis
10. Inactive adult type lesion
11. Decision regarding activity not possible
12. Active adult type lesion
13. Progression
14. Regression
15. Atelectasis, tracheobronchial tuberculosis
16. Pneumothorax
17. Visceroparietal pleural adhesions
18. Pleuritis without effusion
19. Pleural effusion, empyema
20. Cavity
21. Cavity suspected
22. Miliary tuberculosis (Code also 3, 5, 10, 11, or 12)
23. Phrenic interruption
24. Thoracoplasty
25. Other surgical procedure
26. Sinus tract
27. Except as above
29. Unsatisfactory examination
30. Special interest

Examination type

A. Chest, stereo
B. Chest, single film
C. Chest, grid film
D. Chest, lateral or oblique
E. Chest, portable
F. Chest, thymus technic
J. Chest, fluoroscopy

P. Except as above

Follow the diagnosis number by X if the diagnosis has been coded on a previous examination.

Field IX: Gastro-Intestinal Tract

1. Negative (Code examination type)
2. Abnormal (Code examination type. Do not code 2 if examination type is to be coded in another field.)
3. Calcification within abdomen
4. Intestinal obstruction, paralytic ileus
5. Subphrenic or other peritoneal abscess, perforation of hollow viscus
6. Diaphragmatic hernia, eventration of diaphragm, paralysis of diaphragm
7. Extra-alimentary mass involving gut, neoplastic (code also) or inflammatory
8. Extra-alimentary mass not involving gut, neoplastic (code also) or inflammatory
9. Cardiospasm, pylorospasm, irritability of colon
10. Delay in gastric emptying
12. Cholecystogram, faint visualization

13. Cholecystogram, non-visualization
14. Abnormality of liver or spleen
15. Foreign body
16. Ulcer
17. Ulcerative colitis, including tuberculous and regional ileitis and colitis and lymphopathia venereum
18. Diverticulum
19. Calculus
20. Foreign body, soft tissue abnormality
21. Benign neoplasm
22. Malignant neoplasm, primary
23. Malignant neoplasm, secondary
24. Anomaly
25. Postoperative
26. Sinus tract
27. Except as above
28. Diagnosis questionable (Code probable diagnosis also)
29. Unsatisfactory examination
30. Special interest

Examination type

A. Abdomen
B. Complete G.I.; G.B.; colon and stomach
C. Stomach
D. Cholecystogram
E. Colon
G. Cholangiogram

P. Except as above

Localization (*use only when necessary*)

A. Esophagus
B. Stomach
C. Duodenum
D. Biliary tract
E. Colon
F. Small intestine
G. Diaphragm

P. Except as above

Follow the diagnosis number by X if the diagnosis has been coded on a previous examination.

Field X: Genito-Urinary Tract

1. Negative (Code examination type)
2. Abnormal (Code examination type. Do not code 2 if examination type is to be coded in another field.)

Genital Tract

3. Pregnancy
4. Obstetrically abnormal pelvis
6. Pelvic mass, ovarian cyst

Urinary Tract

7. Prostatic hyperplasia
8. Contracted or trabeculated bladder
9. Incomplete filling, pyelogram
10. Absence of kidney
11. Enlarged or contracted kidney
12. Abnormal position of kidney or ureter
13. Hydronephrosis
14. Hydro-ureter
15. Perinephritic abscess
16. Pyelonephritis, pyonephritis
17. Tuberculosis, renal, ureteral
18. Diverticulum
19. Calculus
20. Foreign body, soft tissue abnormality
21. Benign neoplasm
22. Malignant neoplasm, primary
23. Malignant neoplasm, secondary
24. Anomaly
25. Postoperative
26. Sinus tract
27. Except as above
28. Diagnosis questionable (Code probable diagnosis also)
29. Unsatisfactory examination
30. Special interest

Examination type

A. Abdomen
B. Excretory pyelogram
C. Retrograde pyelogram
D. Cystogram
E. Urethrocystogram
F. Uterogram
G. Pelvimetry

P. Except as above

Localization (*use only when necessary*)

A. Genital tract
B. Kidney
C. Ureter
D. Bladder
E. Urethra
F. Prostate

P. Except as above

Follow the diagnosis number by X if the diagnosis has been coded on a previous examination.

CONCLUSIONS

A revision of the Hodges and Lampe cross-index code for radiologic diagnosis is presented. If statistical review is not desired, the system may be simplified by leaving out examination types, normal cases, and follow-up examinations.

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Objectives and Radiological Aspects of Tuberculosis Control Program[1]

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THE CONTROL of tuberculosis has long been a serious medical problem. The problem has been particularly difficult because there is neither an effective immunizing agent against nor a specific cure for the disease. In recent years, efforts have been directed chiefly toward the discovery of the sources of infection and the avoidance of exposure to them. This will continue to be the most important course of action until more decisive methods of control are developed.

It has been recently shown (1) that an effective program of tuberculosis control should embrace four principal phases: (*a*) case-finding, (*b*) medical care and isolation, (*c*) aftercare and rehabilitation, and (*d*) protection of the tuberculous family against economic distress. A program which includes these public-health measures, supported by research and well planned health education in each field of endeavor, will be certain to reduce the morbidity and mortality from tuberculosis.

CASE-FINDING

The purpose of a case-finding program is the discovery of the hidden cases of tuberculosis. In general, such a program should be directed toward those groups of the population where a high prevalence of disease is suspected and where large numbers of the people can be reached quickly and economically. Until recent years case-finding efforts were centered primarily upon the families of known infectious patients. Since tuberculosis is basically a family epidemic, a high yield of new cases was obtained by this approach. However, limited field-nursing services and clinical facilities have greatly restricted the program except in those few communities with well developed and ample health services. Moreover, there are many tuberculous families scattered through the population in which the disease is completely unsuspected. It has been necessary, therefore, to supplement and complement these family epidemiological studies with other case-finding procedures.

Since the introduction of mass radiography, case-finding has been directed on an extensive scale to large population groups without reference to specific foci of infection. This type of program has been so satisfactory that many physicians have advocated that the entire population be examined radiographically at regular intervals. Such a scheme, however, is rather difficult and, furthermore, does not appear to be essential for the control of tuberculosis. As in the control of other communicable diseases, it is probably necessary to reach only a significant proportion of the population within a limited period of time.

There are two sizable segments of the population which may be easily reached by mass radiography, namely persons admitted to general hospitals and persons employed in the large and small industries of the nation.

Small-film radiography is well suited

[1] Presented at the Joint Meeting of the American Roentgen Ray Society and the Radiological Society of North America, Chicago, Ill., Sept. 24–29, 1944.

to case-finding in general hospitals. No expense is entailed in assembling the people for study. In addition, film interpretation may be done by the staff of the department of roentgenology. Furthermore, facilities are already available for completing clinical examinations and providing care and treatment for ambulatory patients. The procedure also provides several valuable by-products. Increased accuracy in the clinical diagnosis of chest disease is obtained. Non-tuberculous disease is detected more quickly than before. Finally, and of particular importance, employees and nurses in contact with patients are spared unnecessary exposure to those who have tuberculosis in a communicable stage.

Hodges (2) at the University of Michigan Hospital, Ann Arbor, Childress *et al.* (3, 4) at Grasslands Hospital, New York, and Bloch and Tucker (5) at the University of Chicago Clinics and Provident Hospital, Chicago, have been examining routinely those admitted to their respective institutions for some time. In Michigan, where the photofluorographic process is employed, 9.3 per cent of the patients present abnormal roentgen findings; about 1.5 per cent exhibit x-ray evidence of pulmonary tuberculosis. In New York, where the fluoroscopic method is used, 2.8 per cent of the patients are found to have reinfection-type tuberculosis. In Chicago, where fluoroscopy and the sensitized paper method are employed, 1.3 per cent of the white patients and 2.64 per cent of the colored admissions give evidence of clinically active tuberculosis. From these figures, the value of mass radiography in the examination of admissions to general hospitals is evident.

It is hoped that soon all general hospitals will provide routine x-ray examinations of the chest just as they are now making routine serologic tests for syphilis. In 1943, over 15 million persons (6), not including outpatients, were admitted to general hospitals in the United States for care and treatment. The newly discovered cases of tuberculosis found among these patients can logically become the centers from which many other cases can be revealed.

The technical problems associated with the development of radiographic case-finding programs in general hospitals need not be difficult. In small hospitals, of less than 200-bed capacity, the number of admissions is sufficiently low to permit the performance of the examinations in the department of radiology itself, without placing an excessive burden on that organization. An inexpensive photofluorographic unit may be installed to utilize the existing x-ray generating equipment or, if the case-load is sufficiently small, examinations may be done with 14 × 17-inch films. In hospitals of greater than 200-bed capacity, a complete photofluorographic installation (with its own x-ray generating equipment) is usually required. In many instances, however, this equipment need not be expensive. Where the case-load is not great, units with hand-operated cameras may be employed. Installations may be either in the department of radiology or adjacent to the admitting office. In large hospitals, completely automatic photofluorographs constituting an integral part of the admitting department are preferable since they permit the examination of large numbers of patients with a minimum amount of effort.

Hospitals which care for the mentally ill are also ideal centers in which to develop mass radiographic methods. In the United States nearly 500,000 patients are currently hospitalized in these institutions. Chest surveys conducted in Minnesota, New York, and Illinois have shown that from 4 per cent to 10 per cent of these patients have x-ray evidence of reinfection tuberculosis. These people are not only likely to infect fellow patients and the institutional members with whom they come in contact but, when released from care, can disseminate their disease to the general population.

The second population group in which mass radiographic procedures may be profitably conducted consists of the millions of industrial workers. From 1942 to 1944,

over one million workers in the United States were examined by eight transportable field units (35-mm. and 4 × 5-inch) of the United States Public Health Service. In this group of adults, 1.5 per cent gave x-ray evidence of reinfection-type tuberculosis. Of these cases approximately 65 per cent were minimal, 30 per cent moderately advanced, and 5 per cent far advanced, according to the classification of the National Tuberculosis Association. This distribution is of considerable interest in view of the fact that minimal cases comprise only 10 to 15 per cent of the first admissions to tuberculosis hospitals in this country in recent years.

Other chest conditions besides tuberculosis were frequently discovered by means of these industrial surveys. One per cent of the films exhibited evidence of nontuberculous pulmonary disease; about one-half of these showed cardiac abnormalities. A number of films gave evidence of unsuspected cancer of the lungs, early enough in many instances for operative intervention. Certainly no industrial hygiene program can be considered complete unless there is included a routine chest x-ray examination of every employee prior to employment and at regular intervals thereafter.

The United States Public Health Service, which has recently been authorized by the 78th Congress to expand its tuberculosis control program, plans to furnish financial assistance to state, city, and other health groups for the establishment of mass radiographic case-finding procedures, among the 15,000,000 persons admitted to general hospitals each year and among the millions of industrial workers; funds for additional clinics, nursing services, and laboratory facilities to complement this work will also be provided. The Public Health Service itself plans to operate throughout the United States only twenty field x-ray units. These units will be used for the purpose of demonstrating mass radiographic technic and will be sent to local communities at the request of state health departments. Each unit will consist of a medical officer, a public health nurse, two x-ray technicians and a record analyst, and complete equipment for mass radiography. It is hoped that these units will set high standards for tuberculosis control, act as local training centers, and in addition give actual service to the community.

In view of the present film shortage, it has frequently been asked what effect a national tuberculosis case-finding program has on current film supplies. A careful investigation reveals that many millions of people may be examined annually by mass radiographic methods (small sized survey films with 14 × 17-inch films in the positive cases) without requiring more than an extremely small fraction of the total civilian medical film production.

MEDICAL CARE AND ISOLATION

From a public-health standpoint, the primary purpose of sanatorium care is isolation of the patient to prevent spread of the disease. In addition, there is the opportunity to arrest the disease if it is not too advanced and to provide general physical restoration of the individual.

The full benefits of early diagnosis of pulmonary tuberculosis can be realized only if an adequate number of hospital or sanatorium beds are readily available for treatment of those with remediable disease and for isolation of the infectious patients. These institutions must be supplemented by well located chest clinics, generous public-health nursing services, accessible laboratory facilities, and the active participation of family physicians.

It is short-sighted to concentrate on case-finding if treatment is to be delayed because of a shortage of sanatorium beds. Once a program is started in a community, immediate plans must be made for providing a sufficient number of beds. Temporary facilities, including beds in general hospitals, should be utilized until tuberculosis hospitals are available.

At present, long periods of hospitalization are usually necessary for the care and treatment of tuberculous patients. As

mass radiography reaches larger numbers of the population, however, shorter periods of care will frequently be the rule, since many of the patients will have less extensive disease. If sufficient clinical facilities are established throughout the country, such persons, including those on collapse therapy, may often be transferred to the chest clinic for treatment and supervision. Others need only enter local convalescent homes.

In those unfortunately situated communities where no sanatoria or tuberculosis clinics are established, an attempt should be made to isolate known open cases in the home until such time as hospital facilities become available. The public-health nurse can play an important role by demonstrating simple methods of isolation and disinfection and by attempting to obtain examination of household contacts at frequent intervals.

AFTER-CARE AND REHABILITATION

Since tuberculosis often causes considerable disability, many persons who contract the disease must make an adjustment in their way of living when released from sanatorium care. This adjustment is not an easy one, and any guidance which can be given during the period of hospitalization will prepare the way for effective rehabilitation.

Soon after the tuberculous patient has entered the sanatorium he should have an opportunity to discuss with his physician the medical problems of his particular case so that an understanding of his condition may be acquired at an early date. As times goes on, further conferences will permit the establishment of an acceptable regime which may be followed not only during the days of hospitalization but in the period thereafter. During these discussions the question of the type of work which will be most suitable for the patient to follow will arise. In these conferences, the services of social workers, educational psychologists, and employment officers are essential in reaching a satisfactory choice. It is desirable that facilities be provided for instruction and training in the selected vocation as soon as the patient is medically and emotionally prepared.

Persons with arrested disease should be returned gradually to full-time activity through increasing part-time or modified employment. The Altro Workshops in New York are good examples of this type of sheltered work. It is necessary to subsidize these workers during the period of partial employment in order to provide the family with sufficient income to subsist. Soviet Russia and Great Britain have also carried out successful schemes for sheltered work, particularly during the critical first two years after discharge from hospital care.

The disposition of patients with infectious tuberculosis of a chronic character presents a special problem. Such persons can be ambulatory and do some work, yet they must remain segregated because of the communicability of their disease. The English have arrived at a partial solution through the Papworth and Preston Hall Village Settlements. In Soviet Russia, so-called night sanatoria provide partial medical supervision and regulated rest periods for these persons. Segregated sections of factories have sometime been designated for the employment of persons with chronically active disease. Small local workshops in connection with well established large sanatoria have also been utilized. These patients, of course, require subsidy to supplement their limited income.

There is still another large group of tuberculous persons who require assistance for after-care. These include those who have received maximum benefits from modern therapy but still have active disease and therefore must remain isolated. These persons can well be segregated in local institutions and rest homes where care can be given at a minimum cost, for the protection of the health of the community.

Fundamentally the after-care and rehabilitation of the tuberculous patient constitute low-cost insurance on the tremendous investment of hospitalization. They are essential parts of a tuberculosis

control program and without them such a program falls short of its goal.

PROTECTION OF THE TUBERCULOUS FAMILY AGAINST ECONOMIC DISTRESS

Tuberculosis is a community disease which is important not only in terms of public health but also from the point of view of national economy. Once the disease becomes advanced, the affected person is often disabled for life and dies a premature death. The family, broken up by long periods of disability or death of the breadwinner, almost inevitably is thrown upon public resources for its support. Accordingly, a sound medical program must be complemented by a generous plan of public assistance, particularly for the families of the tuberculous poor. If this is not done, full benefits will not be realized from the other phases of the program and especially from sanatorium care.

It is no simple matter to provide institutional isolation and treatment for all known infectious cases. The problem of inducing these persons to enter sanatoria and to remain there long enough to arrest their disease or prevent its spread to others is a difficult one. If the breadwinner is the one afflicted, his first concern is for the welfare of his family. Unless some provision is made to avoid economic disaster for dependents, physical disaster will eventually befall the tuberculosis sufferer who refuses to continue hospitalization while his family is in want. A national plan to provide adequate protection against loss of wages during treatment, and for several years after arrest of the disease, is the only logical answer to this problem.

In order to achieve the four principal objectives of tuberculosis control, great assistance can be given by a carefully planned program of research in each of the fields of operation. Careful studies and investigations are indicated in the evaluation of present-day public health methods. Frequent inventories must be taken by state and local health departments to determine whether or not measures employed are actually decreasing mortality from the disease.

The application of new technical developments in mass radiography should greatly simplify the problem of case-finding among the population groups now difficult to reach. Mass radiography will make possible the epidemiological investigation of entire communities where only a small number of families could be studied before. This will give the epidemiologist an opportunity to study fundamental relationships in the evolution of pulmonary tuberculosis on an extensive scale.

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Note on Cholecystography with Priodax: Dosage and Gastro-Intestinal Effects[1]

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IT IS a well established fact that the new cholecystographic medium, β-(4-hydroxy - 3,5 - di - iodophenyl) - α - phenyl-propionic acid (Priodax), is superior to the tetraiodophenolphthalein preparations. Its diagnostic reliability is unchallenged, its yields better roentgenograms, it is comparatively easy to take, and it produces fewer and less severe side-effects than does tetraiodophenolphthalein.

The recorded figures for the frequency of gastro-intestinal side-effects vary considerably. Vomiting is reported in different series as having occurred in 0.08 to 3 per cent, nausea in 8 to 26 per cent, and diarrhea in 3 to 23 per cent and "less than one-third" of the cases (1, 2, 3, 4, 5, 6). These variations might be due to differences in patients at various geographical locations, in the dose administered and method of preparation, and—in the case of nausea and diarrhea—to differences of definition.

This paper deals with the frequency of gastro-intestinal effects as observed in two series of 100 cases each, and with the correlation of gastro-intestinal effects and dosage of the contrast medium.

The first series includes 100 consecutive cases referred for gallbladder examination. The patients were prepared as recommended by the manufacturers of Priodax. The dose administered was 6 tablets for patients up to 150 lb., 9 tablets from 150 to 200 lb., and 12 tablets above 200 lb. If the gallbladder was not visualized, a re-examination was done with 12 tablets for patients up to, and 18 tablets for patients above, 150 lb. In every case, we recorded weight and number of tablets taken, also the number of bowel movements between taking the tablets and examination and the occurrence of vomiting and of persistent nausea. Transitory slight nausea of a few minutes' duration was not recorded. The results of this investigation are shown in Table I (Series I). Seventeen patients in this series reported nausea, but none of them vomited. Forty-four registered a mild laxative effect with two to four bowel movements, and 20 had from five to twelve bowel movements. The average number of bowel movements was 2.8.

The patients were divided into classes according to the number of tablets received per pound. In all classes with from 0.03 to 0.06 tablets per pound (or up to one tablet per 17 lb.) the average number of bowel movements was about the same. A total of 56 patients fell into this range (Series IA in the table); they produced an average of 1.9 bowel movements, and 6 (or 11 per cent) registered nausea. The remaining 44 patients (series IB in the table) had received from 0.061 to 0.104 tablets per pound; they produced an average of 4.0 bowel movements, and 11 (or 25 per cent) experienced nausea. These figures indicate that there is a definite relation between dose administered and gastro-intestinal effects. There seems to be a borderline near the dose of one tablet per 17 lb.

Comparative records of gastro-intestinal effects at the first and second examinations were taken in 10 cases. These patients received a total of 78 tablets for the first examination and produced a total of 25 bowel movements. For the re-examination, a total of 118 tablets was administered, with 35 bowel movements resulting. These figures agree with the results recorded in the foregoing paragraph.

The experiences with the first series of

[1] From the Department of Radiology, Carle Hospital Clinic, Urbana, Ill. Accepted for publication in November 1944.

TABLE I: GASTRO-INTESTINAL SIDE-EFFECTS OF PRIODAX

Dosage of Priodax	Bowel Movements (Percentage of patients with 0 to 5 or more bowel movements)							Nausea	Vomiting
	0	1	2	3	4	5 or more	Average		
Series I (100 cases) 0.030 to 0.104 tablets per pound	10	26	20	11	13	20	2.8	17	0
Series IA (56 cases) 0.03–0.06 tablets per pound	14	37.5	21	11	11	5.5	1.9	11	0
Series IB (44 cases) 0.061–0.104 tablets per pound	4.5	11.5	18	10	17	39	4.0	25	0
Series II (100 cases) 0.036–0.05 tablets per pound	20	30	26	10	5	9	1.9	12	3

patients induced us to reduce the doses of Priodax. The following dosage scheme was given to the drug room:

First examination: 4 tablets for patients up to 100 lb., 5 tablets up to 125 lb., 6 tablets up to 150 lb., and so on in steps of one tablet for 25 lb.

Re-examination: 5 tablets up to 83 lb., 6 tablets up to 100 lb., 7 tablets up to 117 lb., and so on in steps of one tablet for 17 lb.

The gastro-intestinal side-effects were recorded in 100 consecutive cases treated according to the changed dosage scheme. These cases form Series II of the table. As expected, the results obtained in this series agree rather closely with those found in Series IA: an average of 1.9 bowel movements, with nausea in 12 per cent and vomiting in 3 per cent of the cases. The gallbladder shadows obtained in the second series were not quite so dense as those of the first one, but perfectly satisfying for diagnostic purposes.

It is evident that high doses of Priodax produce a high frequency of gastro-intestinal side-effects The way to reduce them as far as possible is to avoid double dosing. By keeping the doses for both first and second examination below one tablet per 17 lb., one can hold gastro-intestinal effects to a low level, which probably could not be lowered appreciably by further reduction of the dose within diagnostic range.

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EDITORIAL

Surveys for Tuberculosis

The recent appropriation by Congress of $10,000,000 for the extension of case-finding and the treatment of tuberculosis and the announcement of the plans of the United States Public Health Service for its use have stimulated discussion of tuberculosis surveys and their effect upon the "socialization" of medicine. The ideas expressed range from inordinate praise and optimism on the one hand to unjust objection and pessimism on the other. Judgment both as to the value of such surveys and their effect upon the "socialization" of medicine should be reserved until many aspects of the subject have been thoroughly considered and evaluated with regard to the whole program.

It seems inconceivable that any one interested in public welfare should object to surveys of the population for tuberculosis case-finding. Yet objections have been raised; some because the objectors do not consider surveys justifiable; some because the objectors fear an entering wedge for the government practice of medicine.

There is some basis for adverse criticism of the present hit-and-miss survey of small groups, but it is a short-sighted criticism. The surveys of school children represent a step in the right direction, but they do not reach the age group where investigation is most important. Surveys of the workers in a few factories are also useful, though they do not involve enough of the population to be really effective. Failure to cover the entire field is not, however, a valid reason for condemning group surveys.

The ideal survey would include all of the population in certain age groups annually. At the present time this seems impossible, because of inadequate equipment and personnel. It is, however, the objective toward which we should work. Reliable authorities have said that if universal surveys could be conducted, and follow-up treatment instituted, tuberculosis could be practically eradicated in ten years. The difficulty in attaining this ideal is not sufficient reason for abandoning it as an objective.

A few metropolitan centers are almost in a position to institute universal surveys. These could be used as test areas, furnishing accurate information as to the cost, the necessary personnel, the apparatus needed, etc. From these trial surveys information as to the requirements for the primary examination and follow-up treatment could be gained. Information thus gathered would be invaluable in setting up survey methods in other areas.

This experimental work must obviously be done by a governmental agency, either Federal or state. In the interests of uniformity and success throughout the country, the Federal Government is the most logical supervising agency. The fear that this will be an entering wedge for governmental medicine is no more real than the fear that the state care of the mentally ill will give rise to governmental medicine. The nature of the disease makes state care of the tuberculous patient necessary. Too few people are able to stand the economic strain of a long period of illness and convalescence. To provide sanatoria for their care, we must look to the government, probably both state and Federal. This means government administration of hospitals and government employment of physicians. It is the only reasonable way to solve the problem. The menace of open tuberculosis in one's next-door neighbor, or in the worker at the next bench, is

too serious to be left to charity or unsystematic medical attention. The chief responsibility of the medical profession is to see that the discovery and treatment of tuberculosis conform to proper medical standards, not to political standards.

Experience has shown that where extensive surveys for tuberculosis have been made, the physician, both general and special, has benefited. Instead of losing patients, he has gained them, not only new patients with tuberculosis, but those with other diseases disclosed by the survey. A survey is not an accurate and complete diagnostic measure. Because of its very nature, it hardly can be. It is, rather, a winnowing process, after which come accurate study and diagnosis of the individual case. This becomes the job of the physician, private for those who can afford his services, public for those who cannot take care of themselves. In either event, there is no financial loss to the physician.

Sometimes the method of paying for local surveys has left an incorrect impression in the mind of the public regarding the cost of a chest film. The patient has been led to feel, because of the low unit cost in a survey, that the normal fee for a single examination of the chest by the private radiologist is unreasonably high. This is unfortunate and has sometimes worked to the disadvantage of the private radiologist. With proper publicity and education and proper methods of financing, this fault can be eliminated. The public is familiar with the reduction in costs brought about by mass production. They will be equally intelligent in realizing that the cost of a chest examination in a mass survey will be less than a private and more personalized study.

Group examinations for tuberculosis are of such fundamental importance to the public and individual health that they are coming in spite of the difficulties they present. Before they are completely satisfactory and successful, many trials and errors must be made. It is the duty of everyone concerned with their development to cooperate in every way to minimize the errors. Healthy development can be seriously hindered by attempts at dictation of policies and procedures by the government, social agencies, or professional groups.

SYDNEY J. HAWLEY, M.D.

RADIOLOGY SECTION CALIFORNIA MEDICAL ASSOCIATION

Dr. James B. Irwin, of San Diego, has been elected Chairman of the Section on Radiology of the California Medical Association, and Dr. Gordon G. King, of San Francisco, Secretary.

THE PHILADELPHIA ROENTGEN RAY SOCIETY

The newly elected officers of the Philadelphia Roentgen Ray Society are: President, Dr. Paul C. Swenson; Vice-President, Dr. Louis Edeiken; Treasurer, Dr. David A. Sampson; Secretary, Dr. Calvin L. Stewart.

Books Received

Books received are acknowledged under this heading, and such notice may be regarded as recognition of the courtesy of the sender. Reviews will be published in the interest of our readers and as space permits.

PULMONARY TUBERCULOSIS IN THE ADULT: ITS FUNDAMENTAL ASPECTS. By MAX PINNER, M.D., Chief, Division of Pulmonary Diseases, Montefiore Hospital for Chronic Diseases, New York; Editor, American Review of Tuberculosis; Clinical Professor of Medicine, College of Physicians and Surgeons, Columbia University, New York. A volume of 579 pages, with 59 illustrations and graphs. Published by Charles C Thomas, Springfield, Ill. Price $7.50.

Book Reviews

SUPERVOLTAGE X-RAY THERAPY: A REPORT FOR THE YEARS 1937–1942 ON THE MOZELLE SASSOON SUPERVOLTAGE X-RAY THERAPY DEPARTMENT, ST. BARTHOLOMEW'S HOSPITAL. By RALPH PHILLIPS, M.S., M.B., F.R.C.S., D.M.R.E., Sir Halley Stewart Fellow, with the technical assistance of G. S. INNES, B.Sc., A.M.I.E.E., Physicist to the Mozelle Sassoon Department. With a Foreword by The Rt. Hon. The Lord Horder, G.C.V.O., M.D., F.R.C.P. A volume of 142 pages, with 95 illustrations. Published for The Sir Halley Stewart Trust by H. K. Lewis & Co., Ltd., London, 1944. Price 16*s.* net.

The author has written a monograph on the experience of St. Bartholomew's Hospital, with a million-volt x-ray machine, from 1937 to 1942, inclusive. It is one of the first books, if not the first, dealing exclusively with supervoltage equipment, physical measurements, and clinical observations.

Considerable space is allotted to a detailed description of the supervoltage x-ray machine designed and installed by Messrs. Metropolitan Vickers Electrical Co., Ltd. Various parts of the x-ray machine are well illustrated with photographs and drawings. The descriptions and illustrations should be worth while for those building supervoltage x-ray machines. The one showing a filament holder carrying six interchangeable filaments is interesting.

The building housing the supervoltage x-ray machine is described and illustrated. It was interesting to note that the walls were so sturdy that little damage was done by bombs dropped nearby.

Physical measurements are illustrated by tables and curves. Isodose curves are given for different qualities of supervoltage radiation.

Clinical investigations to determine the number of roentgens to produce a unit skin dose (U.S.D.) are described. The treatment of various types of lesions is illustrated with corresponding isodose curves showing the percentage of radiation delivered to the tumor-bearing area. The number of patients treated and the corresponding life spans are presented.

In conclusion: The book contains useful information for anyone anticipating supervoltage therapy. The clinical application in the use of isodose curves, number, size and direction of portals with total tumor doses, will be of interest to the general radiologist and worth while for reference.

LES BASES PHYSIQUES ET BIOLOGIQUES DE LA ROENTGENTHÉRAPIE. By PAUL LAMARQUE, Docteur ès Sciences, Professeur à la Faculté de Médecine de Montpellier, Directeur du Centre Régional contre le Cancer. With the collaboration of PIERRE BÉTOULIÉRES, Ancien Interne des Hôpitaux, Chef de Laboratoire de Radiologie à la Faculté de Médecine de Montpellier, JEAN REBOUL, Docteur ès Sciences, Chef de Laboratoire de Radiologie à la Faculté de Médecine de Montpellier, EDMOND DEBAINS, Ingénieur E.P.C.I., and PIERRE LORIMY, Ingénieur E.S.E. Preface by Professeur Strohl, Membre de l'Académie de Médecine. A volume of 530 pages, with numerous illustrations. Published by Masson et Cie, Ed., Paris.

This volume of 530 pages, well printed on good paper, is a comprehensive and competent work. It is well illustrated with good line drawings. The material on basic physics, written in collaboration with Reboul, is handled with a minimum of mathematics, but contains considerable theoretical material, presented in lucid fashion. The section on x-ray tubes and generators, in collaboration with Lorimy and Debains, is quite complete.

The chapter on qualitative measurements of x-rays is excellent, containing data on spectrometry, direct and indirect voltage measurement, and the various schemes dependent on absorption measurements. In the chapter on quantitative measurements and practical dosimetry, most of the standard dosimeters are described in some detail. A good deal of material is given for the determination of the surface dose, but very little for depth doses. Some additional curves and tables are included in a later chapter, but nowhere does one find enough material for practical dosage calculations.

The section devoted to biological effects contains detailed discussions of reversible and irreversible, early and late effects, radiosensitivity, time and intensity and quality factors. Bétoulierès in his chapter, after describing the response to radiation of various tissues and organs, outlines theories of the mechanism of action of radiation. Reboul, in his, goes into considerable detail regarding the various theories of action of a given dose of radiation, first on a biologic population and second on a single cell.

Under therapy technics, a good deal of space is devoted to very low and very high voltages, but little to the region most commonly available. The section on time factor and fractionation of dose should be considered a historical review of the subject; it presents a group of methods, taken from the literature and disagreeing somewhat among themselves, without critical discussion.

The chapters on radiological accidents and protection are thorough, including the French official regulations.

The book concludes with a bibliography of 651 items and an extensive table of contents. Unfortunately there is no index.

RADIOLOGICAL SOCIETIES OF NORTH AMERICA

Editor's Note.—Will secretaries of societies please cooperate by sending information to Howard P. Doub, M.D., Editor, Henry Ford Hospital, Detroit 2, Mich.

UNITED STATES

Radiological Society of North America.—Secretary, D. S. Childs, M.D., 607 Medical Arts Bldg., Syracuse 2, N.Y.

American Roentgen Ray Society.—Secretary, Harold Dabney Kerr, M.D., Iowa City, Iowa.

American College of Radiology.—Secretary, Mac F. Cahal, 20 N. Wacker Dr., Chicago 6, Ill.

Section on Radiology, American Medical Association.—Secretary, U. V. Portmann, M.D., Cleveland Clinic, Cleveland 6, Ohio.

ARKANSAS

Arkansas Radiological Society.—Secretary, J. S. Wilson, M.D., Monticello. Meets every three months and annually at meeting of State Medical Society.

CALIFORNIA

California Medical Association, Section on Radiology.—Secretary, Gordon King, M.D., Children's Hospital, San Francisco.

Los Angeles County Medical Association, Radiological Section.—Secretary, Roy W. Johnson, M.D., 1407 South Hope St., Los Angeles. Meets second Wednesday of each month at County Society Building.

Pacific Roentgen Society.—Acting Secretary, Frederick H. Rodenbaugh, M.D., 490 Post St., San Francisco. Meets annually with California Medical Association.

San Diego Roentgen Society.—Secretary, Henry L. Jaffe, M.D., U. S. Naval Hospital, San Diego, Calif. Meets first Wednesday of each month.

San Francisco Radiological Society.—Secretary, Carlton L. Ould, University Hospital, Medical Center, San Francisco 22. Meets monthly on the third Thursday at 7:45 P.M., first six months of the year in Lane Hall, Stanford University Hospital, and second six months in Toland Hall, University of California Hospital.

COLORADO

Denver Radiological Club.—Secretary, A. Page Jackson, Jr., M.D., 304 Republic Bldg., Denver 2. Meetings third Friday of each month, Denver Athletic Club.

CONNECTICUT

Connecticut State Medical Society, Section on Radiology.—Secretary, Max Climan, M.D., 242 Trumbull St., Hartford 3. Meetings bimonthly, second Thursday.

FLORIDA

Florida Radiological Society.—Secretary-Treasurer, J. F. Pitman, M.D., Blanche Hotel Annex, Lake City.

GEORGIA

Georgia Radiological Society.—Secretary-Treasurer, James J. Clark, M.D., 478 Peachtree St., N. E., Atlanta 3. Meets in November and at the annual meeting of State Medical Association.

ILLINOIS

Chicago Roentgen Society.—Secretary, Fay H. Squire, M.D., 1753 W. Congress St., Chicago 12. Meets at the Palmer House, second Thursday of October, November, January, February, March, and April.

Illinois Radiological Society.—Secretary-Treasurer, William DeHollander, M.D., St. Johns' Hospital, Springfield. Meetings quarterly by announcement.

Illinois State Medical Society, Section on Radiology.—Secretary, Frank S. Hussey, M.D., 250 East Superior St., Chicago 11.

INDIANA

The Indiana Roentgen Society.—Secretary-Treasurer, Harold C. Ochsner, M.D., Methodist Hospital, Indianapolis 7. Annual meeting in May.

IOWA

The Iowa X-ray Club.—Secretary, Arthur W. Erskine, M.D., Suite 326 Higley Building, Iowa City. Holds luncheon and business meeting during annual session of Iowa State Medical Society.

KENTUCKY

Kentucky Radiological Society.—Secretary-Treasurer, Sydney E. Johnson, 101 W. Chestnut St., Louisville.

LOUISIANA

Louisiana Radiological Society.—Secretary-Treasurer, Johnson R. Anderson, M.D., North Louisiana Sanitarium, Shreveport. Meets annually at same time as State Medical Society.

Shreveport Radiological Club.—Secretary, Oscar O. Jones, M.D., 2622 Greenwood Road. Meets monthly September to May, third Wednesday, 7:30 P.M.

MARYLAND

Baltimore City Medical Society, Radiological Section.—Secretary, Charles N. Davidson, M.D., 101 West Read St., Baltimore 1.

MICHIGAN

Detroit X-ray and Radium Society.—Secretary-Treasurer, E. R. Witwer, M.D., Harper Hospital, Detroit 1. Meetings first Thursday of each month from October to May, at Wayne County Medical Society club rooms.

Michigan Association of Roentgenologists.—Secretary, Bruce MacDuff, M.D., 201 Sherman Bldg., Flint 3.

MINNESOTA

Minnesota Radiological Society.—Secretary, A. T. Stenstrom, M.D., Minneapolis General Hospital, Minneapolis 26. Meetings quarterly.

MISSOURI

Radiological Society of Greater Kansas City.—Secretary, John W. Walker, M.D., 306 E. 12th St., Kansas City, Mo. Meetings last Friday of each month.

St. Louis Society of Radiologists.—Secretary, Edwin C. Ernst, M.D., 100 Beaumont Medical Bldg. Meets on fourth Wednesday of each month except June, July, August, and September.

NEBRASKA

Nebraska Radiological Society.—Secretary-Treasurer, Donald H. Breit, M.D., University of Nebraska Hospital, Omaha 5. Meetings third Wednesday of each month at 6 P.M. in either Omaha or Lincoln.

NEW ENGLAND

New England Roentgen Ray Society.—Secretary-Treasurer, George Levene, M.D., Massachusetts Memorial Hos-

pitals, Boston, Mass. Meets monthly on third Friday at Boston Medical Library.

NEW HAMPSHIRE

New Hampshire Roentgen Society.—Secretary-Treasurer, Richard C. Batt, M.D., St. Louis Hospital, Berlin.

NEW JERSEY

Radiological Society of New Jersey.—Secretary, H. R. Brindle, M.D., 501 Grand Ave., Asbury Park. Meetings at Atlantic City at time of State Medical Society and midwinter in Newark as called.

NEW YORK

Associated Radiologists of New York, Inc.—Secretary, William J. Francis, M.D., East Rockaway, L. I.

Brooklyn Roentgen Ray Society.—Secretary-Treasurer, Leo A. Harrington, M.D., 880 Ocean Ave., Brooklyn 26. Meets fourth Tuesday of every month, October to April.

Buffalo Radiological Society.—Secretary-Treasurer, Joseph S. Gian Franceschi, M.D., 610 Niagara St., Buffalo 1. Meetings second Monday evening each month, October to May, inclusive.

Central New York Roentgen Society.—Secretary-Treasurer, Carlton F. Potter, M.D., 425 Waverly Ave., Syracuse 10. Meetings in January, May, and October.

Long Island Radiological Society.—Secretary, Marcus Wiener, M.D., 1430 48th St., Brooklyn 19. Meetings fourth Thursday evening each month at Kings County Medical Bldg.

New York Roentgen Society.—Secretary, Wm. Snow, M.D., 941 Park Ave., New York 28.

Rochester Roentgen-Ray Society.—Secretary, Murray P. George, M.D., 260 Crittenden Blvd., Rochester 7. Meets at Strong Memorial Hospital, third Monday, September through May.

NORTH CAROLINA

Radiological Society of North Carolina.—Secretary-Treasurer, Major I. Fleming, M.D., 404 Falls Road, Rocky Mount. Meets in May, and October.

NORTH DAKOTA

North Dakota Radiological Society.—Secretary, Charles Heilman, M.D., 1338 Second St., N., Fargo.

OHIO

Ohio Radiological Society.—Secretary, Henry Snow, M.D., 1061 Reibold Bldg., Dayton 2. Next meeting at annual meeting of the Ohio State Medical Association.

Cleveland Radiological Society.—Secretary-Treasurer, Don D. Brannan, M.D., 11311 Shaker Blvd., Cleveland 4. Meetings at 6:30 P.M. on fourth Monday of each month from October to April, inclusive.

Radiological Society of the Academy of Medicine (Cincinnati Roentgenologists).—*Secretary-Treasurer,* Samuel Brown, M.D., 707 Race St., Cincinnati 2. Meetings held third Tuesday of each month.

PENNSYLVANIA

Pennsylvania Radiological Society.—Secretary-Treasurer, L. E. Wurster, M.D., 416 Pine St., Williamsport 8. The Society meets annually.

Philadelphia Roentgen Ray Society.—Secretary, Calvin L. Stewart, M.D., Jefferson Hospital, Philadelphia 7. Meets first Thursday of each month at 8:00 P.M., from October to May, in Thomson Hall, College of Physicians, 21 S. 22d St.

Pittsburgh Roentgen Society.—Secretary-Treasurer, Lester M. J. Freedman, M.D., 4800 Friendship Ave., Pittsburgh 24. Meets second Wednesday of each month at 6:30 P.M., October to May, inclusive, at The Ruskin, 120 Ruskin Ave.

ROCKY MOUNTAIN STATES

Rocky Mountain Radiological Society (North Dakota, South Dakota, Nebraska, Kansas, Texas, Wyoming, Montana, Colorado, Idaho, Utah, New Mexico).—*Secretary,* A. M. Popma, M.D., 220 North First St., Boise, Idaho.

SOUTH CAROLINA

South Carolina X-ray Society.—Secretary-Treasurer, Robert B. Taft, M.D., 103 Rutledge Ave., Charleston 16.

TENNESSEE

Memphis Roentgen Club.—Chairmanship rotates monthly in alphabetical order. Meetings second Tuesday of each month at University Center.

Tennessee Radiological Society.—Secretary-Treasurer, J. Marsh Frère, M.D., 707 Walnut St., Chattanooga. Meeting annually with State Medical Society in April.

TEXAS

Dallas-Fort Worth Roentgen Study Club.—Secretary, X. R. Hyde, M.D., Medical Arts Bldg., Fort Worth, Texas. Meetings on third Monday of each month, in Dallas in the odd months and in Fort Worth in the even months.

Texas Radiological Society.—Secretary-Treasurer, Asa E. Seeds, M.D., Baylor Hospital, Dallas.

VIRGINIA

Virginia Radiological Society.—Secretary, E. Latané Flanagan, M.D., 215 Medical Arts Bldg., Richmond 19.

WASHINGTON

Washington State Radiological Society.—Secretary-Treasurer, Thomas Carlile, M.D., 1115 Terry Ave., Seattle. Meetings fourth Monday of each month, October through May, at College Club, Seattle.

WISCONSIN

Milwaukee Roentgen Ray Society.—Secretary-Treasurer, C. A. H. Fortier, M.D., 231 W. Wisconsin Ave., Milwaukee 3. Meets monthly on second Monday at the University Club.

Radiological Section of the Wisconsin State Medical Society.—Secretary, S. R. Beatty, M.D., 185 Hazel St., Oshkosh. Two-day annual meeting in May and one day in connection with annual meeting of State Medical Society in September.

University of Wisconsin Radiological Conference.—Secretary, E. A. Pohle, M.D., 414 N. Charter St., Madison 6. Meets first and third Thursdays, 4 to 5 P.M., September to May, inclusive, Room 301, Service Memorial Institute.

CANADA

Canadian Association of Radiologists.—Honorary Secretary-Treasurer, J. W. McKay, M.D., 1620 Cedar Ave., Montreal.

La Société Canadienne-Française d'Electrologie et de Radiologie Médicales.—General Secretary, Origène Dufresne, M.D., Institut du Radium, Montreal. Meets on third Saturday of each month.

CUBA

Sociedad de Radiología y Fisioterapia de Cuba.—Offices in Hospital Mercedes, Havana. Meets monthly.

ABSTRACTS OF CURRENT LITERATURE

ROENTGEN DIAGNOSIS

The Chest

EDLING, NILS P. G. X-Ray Diagnosis of Retrocardial and Retrodiaphragmatic Infiltrations of the Lungs 200

SEYMOUR, MINER W. Disqualifying Pulmonary Defects in 100,000 Selectees Examined Radiographically by an Armed Forces Induction Station 200

GUNDERSEN, SVEN. Primary Atypical Pneumonia of Unknown Etiology 200

CARSON, M. J., AND WEBB, A., JR. Congenital Lung Cyst Complicated by Pyopneumothorax 200

BARRETT, N. R. Lung Abscess. Pathology and Diagnosis of Certain Types 201

BILLING, L. Unusual X-Ray Findings in a Case of Pedunculated Pulmonary Fibroma 202

QUINN, ROBERT W. Health Hazards Associated with the Welding Process. Report of Cases and Review of Recent Literature 202

MIMPRISS, T. W., AND ETHERIDGE, F. G. Post-Operative Chest Complications in Gastric Surgery 202

HURST, ALLAN, AND BASSIN, SYDNEY. Mega-Esophagus as a Cause of Mediastinal Widening 202

DERBES, V. J., AND ENGELHARDT, H. T. Roentgen Studies of the Heart in Asthmatic Children 202

RITVO, MAX, AND VOTTA, PAUL J. Clinical and Roentgen Manifestations of Dissecting Aneurysm of the Aorta 203

KINZER, RICHARD E., AND COOK, JAMES C. Lesions of the Diaphragm, with Special Reference to Eventration and a Report of Three Cases 203

The Digestive System

MCCLINTON, JAMES B. Non-Malignant Duodeno-Colonic Fistula 203

NØRGAARD, FLEMMING. Roentgenologic Diagnosis of Gallstone Ileus, Specially as Regards the Demonstration of Obstructing Radiotransparent Concrements 203

OPPENHEIMER, ALBERT. Roentgen Diagnosis of Incipient Cancer of the Rectum 203

HELLMER, HANS. Contours of the Right Lobe of the Liver in Ascites 203

METHENY, DAVID, ROBERTS, EDWARD W., AND STRANAHAN, ALLAN. Acute Pancreatitis with Special Reference to X-Ray Diagnosis 204

PINKHAM, ROLAND D. Pancreatic Collections (Pseudocysts) Following Pancreatitis and Pancreatic Necrosis: Review and Analysis of Ten Cases 204

The Peritoneum

STEINBERG, BERNHARD, AND MARTIN, RUTH A. Diffusion and Localization of Experimental Infections of the Peritoneum 204

The Spleen

NEIDHARDT, H. W. Solitary Calcified Cyst of the Spleen. Report of a Case 205

The Skeletal System

COBURN, DONALD E. Severe Osteitis Fibrosa Cystica with Parathyroid Tumor. Report of a Case of Fifteen Years' Duration 205

HAGLUND, FRED. Importance of Degeneration of the Cervical Disks for the Narrowing of the Intervertebral Foramina 206

WALKER, A. EARL. Dilatation of the Vertebral Canal Associated with Congenital Anomalies of the Spinal Cord 206

CRAMER, FRITZ, AND MCGOWAN, FRANCIS J. Role of the Nucleus Pulposus in the Pathogenesis of So-Called Recoil Injuries of the Spinal Cord 206

COHN, BERNARD N. E. Painful Shoulder Due to Lesions of the Cervical Spine 207

HOLM, OLOV FR. Etiology of Phosphorus Sclerosis 207

JÖNSSON, GUNNAR. Aseptic Bone Necrosis of the Os Capitatum (Os Magnum) 207

DEW, WALTER A., AND WOOTEN, JAMES H., JR. March Fractures, a Series of Fifty-Eight 207

Gynecology and Obstetrics

SOHRNE, G. Roentgenologic Diagnosis of Placenta Previa 207

The Genito-Urinary Tract

SENGER, FEDOR L., AND BOTTONE, JOHN J. Perirenal Insufflation 207

LEVANT, BENJAMIN, AND YARDUMIAN, KRIKOR. Reflex Anuria Following Retrograde and Excretory Investigation 207

GOLDSTONE, B. W. Method of Locating a Mobile Renal Calculus at Operation 208

NICHOLS, RALPH W., AND LOWMAN, ROBERT M. Patent Urachus 208

RADIOTHERAPY

Neoplasms

WARD, ROBERTSON. Malignant Goiter 208

HAAGENSEN, CUSHMAN, D., AND STOUT, ARTHUR P. Synovial Sarcoma 209

BARRETT, CHANNING W. The Place of Surgery in Fibroids of the Uterus 210

DEAKINS, MARTIN L., FRIEDGOOD, HARRY B., AND FERREBEE, JOSEPH W. Some Effects of Testosterone, Testosterone Propionate, Methyl

Testosterone, Stilbestrol, and X-Ray Therapy in a Patient with Cushing's Syndrome.. 210

Non-Neoplastic Diseases

SAGHER, FELIX. Effect of Grenz Rays on Leprous Infiltrations: Report of an Attempt to Influence Leprous Infiltrations by Roentgen Rays of Long Wavelength..... 210

FOWLER, EDMUND P., JR. Use of Radon to Prevent Otitis Media Due to Hyperplasia of Lymphoid Tissue and Barotrauma (Aero-Otitis)..... 210

Radiation Effects

MCINTOSH, H. C., AND HUTTON, J. E. Clinical and Roentgen Aspects of Irradiation Stricture of the Rectum and Sigmoid: Its Course and Treatment..... 211

GLASS, S. J. Dwarfism Associated with Microcephalic Idiocy and Renal Rickets..... 211

TRUELSEN, FRITHIOF. Injury of Bones by Roentgen Treatment of Cancer of the Uterine Cervix..... 211

VAN DER WERFF, J. TH. Mathematical Theory of Biologic Reaction Phenomena, Especially Following X-Ray Irradiation..... 212

HOECKER, FRANK E. Radon/Carbon Dioxide Ratio as an Index to Excessive Radium Absorption..... 212

HOECKER, FRANK E. Significance of Absorbed Radium as Indicated by the Coefficient of Elimination..... 212

VAN GRIETHUYSEN, B. G., AND DEN HOED, D. Supervoltage Roentgen Installation in the Antoni van Leeuwenhoekhuis. Action of Ultra-Hard Roentgen Rays on the Human Skin and on Mice..... 212

THE CHEST

X-Ray Diagnosis of Retrocardial and Retrodiaphragmatic Infiltrations of the Lungs. Nils P. G. Edling. Acta radiol. **23:** 595–602, December 1942. (In German.)

The author discusses the roentgen diagnosis of retrocardiac and retrodiaphragmatic densities of the lungs, proceeding from a study of cases of bronchopneumonia.

In technically perfect frontal roentgenograms retrocardiac shadows may be observed. Retrodiaphragmatic shadows can be seen in lateral or oblique views or in posterior-anterior views if the beam is directed tangentially toward the posterior part of the diaphragmatic dome. The importance of studying the contour of the pneumo-pleural demarcation line in order to detect slight densities is stressed. A break in the continuity of this line is pathologically significant.

MAX SGALITZER, M.D.

Disqualifying Pulmonary Defects in 100,000 Selectees Examined Radiographically by an Armed Forces Induction Station. Miner W. Seymour. Ohio State M. J. **40:** 1041–1045, November 1944.

The findings in 100,000 chest roentgenograms of recruits from southern Ohio are summarized. The total number of men rejected in this series for pulmonary conditions was 2,505 (2.4 per cent); 2,002 (2 per cent) of this number had tuberculous lesions. Sixty-four men (0.06 per cent) had what was considered a possible active primary focus; 427 (0.43 per cent) had stable primary residuals which were too extensive for induction under existing regulations. One per cent of the men examined had reinfection phase tuberculosis in some stage; 0.87 per cent had minimal lesions whose activity was undetermined, and 0.13 per cent advanced disease with cavitation or honeycombing. The three leading non-tuberculous conditions necessitating rejection were bronchiectasis (128), thickened pleura (95), and silicosis (68).

The first 68,045 roentgenograms included in this study were taken on 14 × 17-in. single paper film. The film was read wet by reflected light arranged so as to prevent glare and surface reflection. A tube-screen distance of 48 inches was used, with the following technical factors: 100 ma., 70–90 kv.p., and 0.1–0.15 sec. This gave good penetration and contrast for all except the heaviest men. The remaining 31,995 films were taken by a photoroentgen unit as 4 × 5-in. stereopictures on 4 × 10-in. film. Of several technics, the one finally adopted was as follows: 36-in. tube-screen distance, 150 ma., 80–100 kv.p., 0.2 to 0.3 sec. Any extremely thick man (over 28 cm.) was x-rayed on 14 × 17-in. single film to conserve the tube life of the photoroentgen unit. Retakes with the paper film averaged 3 per 100; with the stereoscopic films, 2 per 100. Approximately 125 to 140 men per hour could be examined with the first method; 65 to 75, with the latter. The diagnostic value of both types of film is good, although the stereoscopic image has some advantage over the flat plate. The paper film has the disadvantage of being bulky to file.

Primary Atypical Pneumonia of Unknown Etiology. Sven Gundersen. New England J. Med. **231:** 697–700, Nov. 23, 1944.

This is a review of 122 carefully studied cases diagnosed as atypical pneumonia. The condition is characterized by the influenza-like symptoms of mild upper respiratory tract infection, with few physical findings, which are slow in appearing and mild as compared with the roentgen findings.

The therapy is symptomatic. Sulfonamides do not influence the course of the disease. Roentgen therapy is advised, but was not used in this series.

Two cases terminated fatally and came to autopsy and the findings are described.

The possibility of confusion with tuberculosis is considered and cautioned against.

JOHN B. MCANENY, M.D.

Congenital Lung Cyst Complicated by Pyopneumothorax. M. J. Carson and A. Webb, Jr. J. Pediat. **25:** 443–446, November 1944.

A case of congenital lung cyst complicated by pyopneumothorax is reported in a 2 1/2-month-old Negro male, who had been well until approximately thirty minutes before admission. The infant was well developed and well nourished but in obvious respiratory distress, with a rapid, grunting type of respiration. The right chest appeared larger than the left and showed a definite respiratory lag. The heart was shifted to the left, the rate was rapid, and the rhythm regular. The percussion note over the entire right chest was hyperresonant and the breath sounds here were extremely faint. A spontaneous pneumothorax was believed to be present, and sulfadiazine was given prophylactically. A roentgenogram two days later showed a right-sided pneumothorax, with the heart shifted to the left, evidence of fluid at the base, and a large cavity at the right apex, apparently in the substance of the lung. During the next three days the infant seemed to improve, but on the fourth day, he suddenly began to breathe with great difficulty, went into shock, and became stuporous. A thoracentesis was immediately performed. A fair amount of air, under high pressure, was removed, together with 40 c.c. of straw-colored fluid, which on culture revealed hemolytic *Staphylococcus aureus*. Respiration ceased and stimulants and artificial respiration were required. Blood transfusions and fluids were then given, and the sulfadiazine increased. On the sixth day, 40,000 units of *Staphylococcus* antitoxin were given intramuscularly, followed by 20,000 units the next day, and improvement was noticeable. A second thoracentesis yielded thick bloody pus, from which hemolytic *Staphylococcus aureus* was again isolated. On the eleventh day, a closed drain was inserted in the right chest between the sixth and seventh ribs in the posterior axillary line and thick pus was obtained. Irrigations were carried out twice daily. Following this there occurred a steady decrease in the amount of drainage, with an increase in appetite, a gain in weight, and some clearing of the chest as shown by roentgenograms and physical examination. Sulfadiazine was discontinued one month after admission, and two days later the patient began to cough persistently and to vomit frequently, the vomitus occa-

sionally containing blood-streaked purulent material. A tentative diagnosis of bronchopleural fistula was made; a rib resection was performed and open drainage of the right chest instituted. Following this, there was rapid improvement, with gradual diminution of the size of the cavity and of the amount of drainage. The patient was discharged on the eighty-second hospital day. A roentgenogram of the chest, taken approximately three months after the onset of the illness, showed practically complete healing and closure of the empyema cavity. The cyst was still prominent at the right apex, apparently unchanged in size or shape.

Lung Abscess. Pathology and Diagnosis of Certain Types. N. R. Barrett. Lancet **2**: 647–651, Nov. 18, 1944.

Five varieties of lung abscess which are sufficiently true to type to warrant separate consideration are described.

Solitary Putrid Lung Abscess. Putrid abscess is the result of the inhalation of infected matter into the terminal bronchioles of a bronchopulmonary segment, the direct cause being certain anaerobic organisms in the bronchial embolus. Once infected material has become impacted in the terminal radicles of the bronchial tree, there is a latent period before the disease is manifest. The lesion which develops in the lung is one of acute infective gangrene. At first it involves a part of one bronchopulmonary segment; in this limited area the bronchioles and their accompanying blood vessels are destroyed and the intervening tissues are liquefied. The inflammation fans out toward the surface of the lobe and the pleura is soon involved, thus producing adhesions over the abscess which prevent its rupture into the pleural space. By about the tenth day a solitary cavity has formed, with an average diameter of two inches. This cavity is peculiar in that it contains impacted sloughs of lung, as well as foul pus, debris, and organisms; its walls are granulation tissue forming a line of demarcation exactly like that seen in gangrene of the extremities. Beyond the cavity the whole of the bronchopulmonary segment concerned is in a state of acute vascular engorgement and edema.

The onset of the illness is sudden, resembling an attack of influenza with pleural pain. Within a few days the picture is more like that of lobar pneumonia. During the first seven to ten days there is little cough or sputum, because the lesion has not yet excavated, but about this time the patient may notice a bad taste in the mouth and perhaps a little blood-stained expectoration. Suddenly, the sputum becomes purulent, abundant, and grossly offensive. Roentgenograms taken about the end of the second week of the illness may show a cavity with a fluid level in an area of consolidation. Such a cavity can be seen in only half of the cases, and failure to demonstrate it roentgenographically does not rule out its presence.

Death is unusual in the first fortnight of the illness, but occurs if the gangrenous process is not localized. As a rule, subsequent events are governed by the establishment, or not, of bronchial channels adequate to drain away the sloughs and other cavity contents. In a third of all cases such drainage takes place spontaneously. Reliable statistics show (1) that it is impossible to predict which acute abscesses will resolve spontaneously, except by observing the lesion for a short period; (2) that the majority of spontaneous cures occur within three months of the onset of symptoms; (3) that the mortality, with treatment of any kind, increases as time passes.

Aerobic Lung Abscess. The first pathological change in the lungs in aerobic abscess is a diffuse bronchopneumonia of unusual type. Small foci of consolidation appear over a wide area in contrast with the early segmental distribution of putrid abscess. As time passes, some of the consolidated areas resolve, some become organized and may be replaced by fibrous tissue, and some suppurate and progress to abscess formation. The foci of pneumonia may be bilateral and spread slowly from one part of the lung to another, so that healing and progress are present together, but there is a general tendency for the lesions to remain in a state of non-resolution. The abscesses are at first small and scattered; the majority discharge their quota of pus into the bronchial tree and heal spontaneously, but in some cases the lesion is more circumscribed, the peribronchial collections of pus coalesce, and large abscess cavities are formed. These are generally loculated and of more irregular shape than putrid abscesses, but clinically differentiation may be impossible. The cause of aerobic abscess is not known, but the organisms in the sputum and in the lung cavities are not anaerobes.

The onset of symptoms is often rather insidious, with lassitude, dyspnea on exertion, an unproductive cough, and pyrexia. The physical signs and the early x-ray findings are typical of bronchopneumonia, but the unusual course of events may suggest pulmonary tuberculosis or neoplasm. The fact that the sputum is not foul is regarded by many as the most significant difference between aerobic and putrid abscess, but the latter may be so well shut off from the bronchial tree that the patient may not discern the characteristic odor. The course of aerobic abscess is rather protracted, but the outlook is better than in putrid abscess. The abscess is merely an incident in the course of a peculiar type of bronchopneumonia and, as such, its presence is relatively unimportant. Surgical measures, such as drainage, are not likely to produce the same dramatic amelioration of symptoms as in putrid abscess.

Staphylococcal abscesses warrant separate consideration because they are often blood-borne, or at least associated with a septicemia, may be affected by such substances as penicillin, and rarely, if ever, require surgical drainage. A staphylococcal abscess may develop either as a complication of staphylococcal pneumonia or as part of a pyemia or septicemia. In both varieties there are pleural complications of great interest and importance. Small cavities situated upon the surface of the lung are apt to perforate into the pleural space and produce spontaneous pneumothorax, tension pneumothorax, and empyema.

The diagnosis of staphylococcal pulmonary abscess depends chiefly upon demonstrating the organism in pure culture in the blood, or in practically pure culture in the sputum, the abscess cavity, or the pleural fluid. The x-ray appearance of the lesions in the chest is so characteristic that many consider it pathognomonic. The opacities are widespread and dense, with rather indefinite outlines: they are often disposed throughout the lungs, and their size and extent immediately suggest a hopeless prospect. Rapid resolution can take place, however. Large cavities are often seen; they are generally empty, or at best partly filled with fluid. During the period of resolution the cavities may persist for a long time as "ring shadows" and can be mistaken for cysts.

Bronchial Abscess. The term bronchial abscess is used to describe a lesion which arises acutely as a result of bronchial occlusion by a relatively large foreign body, such as a tooth. Atelectasis occurs in the affected segment and the bronchi peripheral to the block are at first reduced in caliber. Within a short time mucus and inflammatory fluid collect within these bronchi and infection produces a purulent exudate. It is this intrabronchial collection of pus which is called a "bronchial abscess." If the foreign body is removed, the process settles down and the tissues return to normal. The x-ray appearance is that of atelectasis; the foreign body may or may not be apparent.

Suppurating hematoma of the lung is not well defined but for practical purposes a hematoma is an extravasation of blood of such an extent that it can be diagnosed radiologically. The lesion may result from crush injuries, blast injuries, and penetrating wounds, the blood disrupting the interstitial tissues and lying in the alveoli and bronchi. Organization occurs without cavity formation in most cases and the affected area passes through a phase in which the macroscopic appearance is identical with the gray hepatization of lobar pneumonia. Microscopy, however, shows that the predominating cells are monocytes and not polymorphonuclears. If infection takes place at this stage, a lung abscess forms. The diagnosis is confirmed by blood-stained purulent sputum and by a roentgenogram showing a cavity with a fluid level. The great majority of suppurating hematomas resolve spontaneously and healing is complete.

Unusual X-Ray Findings in a Case of Pedunculated Pulmonary Fibroma. L. Billing. Acta radiol. **23:** 592–594, Dec. 15, 1942. (In German.)

The author reports the x-ray findings in a case of pulmonary fibroma, the size of a child's head, fixed by a peduncle to the left upper lobe of the lung. On changes in the position of the patient, the tumor showed marked motility within the pleural cavity.

MAX SGALITZER, M.D.

Health Hazards Associated with the Welding Process. Report of Cases and Review of Recent Literature. Robert W. Quinn. Mil. Surgeon **95:** 410–419, November 1944.

Six patients with similar acute symptoms of short duration, in an East Coast Navy Yard, were all found to have been exposed to welding fumes, and a diagnosis of metal fume fever was made. The author gives a brief account of his observations and reviews the entire subject of welding injuries, presenting a comprehensive bibliography. X-ray changes may occur in the lungs, closely resembling those of silicosis [see, for example, abstracts of papers by Jones and Lockhart and Sander in Radiology **43:** 405, 1944], but no roentgen studies seem to have been made in the author's patients.

Post-Operative Chest Complications in Gastric Surgery. T. W. Mimpriss and F. G. Etheridge. Brit. M. J. **2:** 466–468, Oct. 7, 1944.

The author presents an interesting discussion of chest conditions following gastric surgery. The gastric lesions are grouped under four headings: (1) ulcers without previous surgery; (2) ulcers with previous ineffective surgery; (3) acute gastric hemorrhage; (4) gastric carcinoma. The pulmonary complications described include acute bronchitis, lobar atelectasis, and lobular or patchy atelectasis. Of a series of 34 cases, 29 were in the last class and 4 of these ended fatally. These 4 cases are reviewed in detail.

The chief etiologic factor in chest complications of gastric surgery is severe peritoneal inflammation. Other factors, *i.e.*, the postoperative condition of the patient and sex and seasonal incidence, are discussed. The principal preventive measure is proper aeration of the lungs by respiratory activity and movement of the patient postoperatively. Where inflammation of the lung has already set in, the usual course for bronchitis is advised.

Q. B. CORAY, M.D.

Mega-Esophagus as a Cause of Mediastinal Widening. Allan Hurst and Sydney Bassin. Am. J. Roentgenol. **52:** 598–606, December 1944.

As a result of routine mass surveys of the chest done primarily for the detection of pulmonary tuberculosis, numerous other lesions are being found, the discovery of which may result in corrective measures of benefit to the individual. One of these lesions is mega-esophagus or cardiospasm. Eleven cases are reported in which this condition was discovered on routine chest roentgenograms and later proved by barium-meal studies. The significant changes which lead to a diagnosis of mega-esophagus are as follows:

(1) Diffuse widening of the mediastinum, usually beginning at the thoracic inlet or just below.

(2) Fluid level. This was seen in 4 of the authors' 11 cases at the level of the clavicles. Its presence depended upon recent eating, drinking, and swallowing of air.

(3) Stippling. The upper part of the shadow may show a stippled or mottled appearance due to a mixture of air and food in the dilated esophagus.

(4) Apparent widening of the cardiac contour due to protrusion of the esophagus beyond the right border. With greater penetration a double margin may be seen.

(5) Obliteration of the cardiophrenic angle.

When these signs are present, mega-esophagus should be suspected and complete barium meal studies should be done.

L. W. PAUL, M.D.

Roentgen Studies of the Heart in Asthmatic Children. V. J. Derbes and H. T. Engelhardt. J. Pediat. **25:** 394–399, November 1944.

Physical and x-ray examinations were carried out in 12 asthmatic children to determine the presence or absence of cardiac hypertrophy. The standard methods of estimating the heart size from roentgen studies were employed. After a preliminary fluoroscopy, two meter plates were taken. The transverse, longitudinal, and broad diameters of the heart and the fluoroscopic observations were within normal limits in each case. Prediction formulae for the transverse diameter of the heart using height and weight ([*a*] $T = 0.0637W + 7.5$; [*b*] $T = 0.00490H + 0.05694W + 7.1$) were worked out, and the results showed a close correlation with the actual measurements. With but three exceptions, the actual transverse diameter was less than the predicted diameter, and in these the difference was minimal. The transverse diameter obtained by the cardiothoracic ratio of Danzer was well within normal limits in each case. The figures for the cardiac area as determined by the Eyster-Hodges nomogram and by computation were, for the most part, in close agreement.

The authors conclude that uncomplicated bronchial asthma is not a factor in heart disease. This does not militate against the production of heart disease subsequent to pulmonary fibrosis, emphysema, bronchiectasis, and other pulmonary complications seen in the chronic asthmatic state.

Clinical and Roentgen Manifestations of Dissecting Aneurysm of the Aorta. Max Ritvo and Paul J. Votta. Am. J. Roentgenol. **52:** 583–594, December 1944.

A dissecting aneurysm is not a true aneurysm in that the lumen of the vessel is not increased. It is produced by penetration of the circulating blood between the layers of the wal of the vessel and is usually associated with a tear in the intima of the artery, hypertension, and atherosclerosis.

Dissecting aneurysm of the aorta is rarely diagnosed during life. Not infrequently death supervenes before adequate studies can be carried out. In the acute cases there is usually a sudden onset with severe pain in the chest or abdomen. The pain may radiate rapidly as dissection progresses. Collapse, shock, and death may occur immediately or be delayed for weeks or months. In these latter cases roentgen examination may be of considerable value. The most significant finding is widening of the supracardiac area. The increase may be to either or both sides. A sudden increase in width of the aortic shadow roentgenologically is diagnostic of dissecting aneurysm. Extravasation of blood into the pericardial sac or pleural cavities may occur, and the roentgen signs of pericardial and pleural effusion may then be elicited. Cardiac hypertrophy is frequently present.

Twelve case reports are included.

L. W. PAUL, M.D.

Lesions of the Diaphragm, with Special Reference to Eventration and a Report of Three Cases. Richard E. Kinzer and James C. Cook. Am. J. Roentgenol. **52:** 611–614, December 1944.

In a series of 412,149 chest roentgenograms made at an Armed Forces Induction Station, 38 examples of diaphragmatic lesions were found. Of these, only three were considered to be true diaphragmatic hernias. The remaining 35 cases were instances of eventration of the diaphragm, 30 occurring on the left side and 5 on the right. Eventration is generally considered to be a congenital condition, since it has been found in the newborn. Pathologically, degeneration of the phrenic nerve secondary to trauma, tumor growth, and cervical tuberculosis have been reported, and it may be that other cases represent a birth injury to the phrenic nerve rather than a true congenital anomaly.

The diagnostic roentgen signs of eventration are a high position, a regular arched contour, and possible changes in excursion due to the defective musculature. The differential diagnosis between eventration and hernia may be difficult and at times impossible.

Three of the cases of right-side eventration are reported briefly. In two of them gas-filled loops of intestine were visible in the lower right thoracic cavity and barium studies revealed parts of the stomach, colon, and small intestine within the space between the diaphragm and liver.

Although most cases of eventration are asymptomatic, the abnormality becomes of clinical significance when it results in gross displacement of abdominal organs.

L. W. PAUL, M.D.

THE DIGESTIVE SYSTEM

Non-Malignant Duodeno-Colonic Fistula. James B. McClinton. Canad. M. A. J. **51:** 434–436, November 1944.

A case of benign duodeno-colonic fistula, diagnosed preoperatively with the aid of a barium enema, is presented. Only four other cases of this condition have been recorded in the literature since 1885 (see McPeak: Radiology **34:** 343, 1940). All cases followed rupture of a duodenal ulcer without operation.

Roentgenologic Diagnosis of Gallstone Ileus, Specially as Regards the Demonstration of Obstructing Radiotransparent Concrements. Flemming Nørgaard. Acta radiol. **23:** 622–628, Dec. 15, 1942. (In English.)

The author reports 3 cases of gallstone ileus in which the diagnosis was established by roentgen examination prior to operation. Although the obstructing concrements in two of these cases were radiotransparent, the diagnosis was made without the use of contrast substances, from a filling defect in the pathologically gas-filled portion of the intestinal lumen. In all cases the obstructing gallstones were shown by the roentgenograms (and found at operation) to lie in the distal ileum.

MAX SGALITZER, M.D.

Roentgen Diagnosis of Incipient Cancer of the Rectum. Albert Oppenheimer. Am. J. Roentgenol. **52:** 637–646, December 1944.

The usual barium enema examination has not been satisfactory for the detection of rectal cancer except in its advanced stages. The author has modified the enema examination so that better demonstration of rectal lesions can be obtained. A flexible urethral catheter is inserted into the rectum and a small quantity of opaque medium is injected under roentgenoscopic control. In most cases 20 to 60 c.c. are sufficient for adequate visualization of the rectum and sigmoid. In addition to spot films, roentgenograms may be made in various positions. After study of the rectum and sigmoid is completed, the rest of the colon is filled in the usual manner.

By this method small lesions in the rectum can be identified. Early cancer of the rectum shows one constant sign, a small ulcer with rigid walls. Filling defects and shelf formation are rarely present. With massive filling the lesion is easily obscured. As the tumor grows, constriction becomes evident, and there may be nodular prominences encroaching upon the lumen.

Carcinomatous polyps, when single, are difficult to see roentgenologically. They produce rounded filling defects as elsewhere in the gastro-intestinal tract. Polyps should be searched for since they may cause no symptoms and a majority will contain foci of carcinoma histologically.

L. W. PAUL, M.D.

Contours of the Right Lobe of the Liver in Ascites. Hans Hellmer. Acta radiol. **23:** 533–540, Dec. 15, 1942. (In German.)

The author shows that in patients suffering from ascites, the lateral contours and parts of the inferior contours of the right lobe of the liver can be demonstrated in roentgenograms of the right upper quadrant without any application of contrast medium to the hepatic parenchyma. By comparison of the different widths

of the interspace between liver and thoracic cage in serial roentgenograms, conclusions are possible regarding the progress of the disease.

MAX SGALITZER, M.D.

Acute Pancreatitis with Special Reference to X-Ray Diagnosis. David Metheny, Edward W. Roberts, and Allan Stranahan. Surg., Gynec. & Obst. **79:** 504–508, November 1944.

The authors report 10 cases of acute pancreatic edema and 17 cases of acute pancreatic necrosis proved at operation or autopsy, together with 5 cases in which the diagnosis of pancreatitis was made on clinical and laboratory findings.

The roentgen findings suggestive of acute pancreatitis are: (1) tender tumefaction of the pancreas found on fluoroscopy, (2) changes in the stomach and duodenum, (3) evidence of localized or generalized ileus. The duodenum shows ileus and loss of tone and on fluoroscopy barium can be pushed into it but fails to move on by peristalsis. The duodenal loop is enlarged. Occasionally enough gas is present in the stomach so that elevation and flattening of the greater curvature can be shown. Increased density of the pancreatic area and an increased space between the greater curvature and transverse colon may also be demonstrable. More frequently a fair-sized "blob of gas" is present in the cardia and a smaller one in the duodenal bulb. Localized ileus, especially of the transverse colon or upper loops of jejunum, occurs. These changes are often evident on a plain scout film of the abdomen in the supine position. The roentgen findings persist as long as the disease lasts.

All the authors' cases had an acute onset with nausea, vomiting, and epigastric pain, but tenderness was often minimal. The temperature and blood pressure were normal and the pulse slow in early cases. Albuminuria was invariably present. The patients with pancreatic necrosis were sicker than those with pancreatic edema and showed a higher total leukocyte count and a higher percentage of polymorphonuclears.

Of the 17 patients with pancreatic necrosis, 10 showed cyanosis, 9 distention, 4 rigidity and tenderness, 4 glycosuria, and 2 jaundice with associated cholelithiasis. The blood amylase remained elevated as long as the disease lasted in the cases of pancreatic edema but returned to normal within two days in the cases of necrosis.

Seventeen patients were treated surgically by various procedures, including cholecystostomy, cholecystectomy, choledochostomy, and gastroenterostomy. Of the 9 patients with pancreatic edema who were operated upon, all survived. Three of the 8 with pancreatic necrosis who were treated surgically survived. The authors believe that survival is more dependent upon the benignity of the disease than upon the type of surgical procedure. For the acute phase of the disease they consider non-operative measures preferable.

One patient with a typical clinical picture of acute pancreatic necrosis and suggestive x-ray findings was given supportive therapy and a small therapeutic roentgen dose. She improved rapidly, and the roentgen findings disappeared. Fourteen days later she was operated on. Stones were found in the gallbladder and ducts and the pancreas was slightly indurated.

Roentgenograms from 9 cases are reproduced.

FRANK P. BROOKS, M.D.

Pancreatic Collections (Pseudocysts) Following Pancreatitis and Pancreatic Necrosis: Review and Analysis of Ten Cases. Roland D. Pinkham. Surg., Gynec. & Obst. **80:** 225–235, March 1945.

The author reviews the literature and speculates on the formation of pancreatic collections (pseudocysts) following pancreatitis and pancreatic necrosis. He points out that the pseudocysts may be found within the substance of the pancreas as the result of degenerative changes affecting the interstitial tissues and parenchyma of the gland. The three common sites are listed as (1) between the stomach and the transverse colon, under the gastrocolic ligament; (2) between the stomach and the liver; (3) between the leaves of the transverse mesocolon.

The relation of acute and chronic pancreatitis to the production of pancreatic cysts and suppurations is well established. Following the first episode of pancreatitis, the pancreas appears to be more vulnerable to recurrent insults. Since there seems to be no infallible way to differentiate, at laparotomy or necropsy between hematogenic collections, pseudocysts, and pancreatic suppurations, the classification and diagnosis of these accumulations are subject to wide individual interpretation. It appears logical to disregard the term "false" or "pseudo" cysts, and designate these as pancreatic cysts or collections resulting from trauma (direct or indirect), duct obstruction (calculi, tumor, inflammation), acute and chronic pancreatitis (infectious, chemical), and pancreatic necrosis (infectious, chemical, and vascular). The term "retention" cysts should be used to designate ductal dilatation arising secondary to partial or complete occlusion of the pancreatic ducts, whereas "true" cysts should be limited to those of a proliferative, hydatid, dermoid, or congenital origin.

The author discusses the significance of a high serum amylase; also when the cyst content contains a high concentration of amylase or any trypsin the diagnosis of pancreatic origin is assured.

Ten cases are reported: 6 were collections subsequent to pancreatic necrosis; 2 subsequent to suppurative pancreatitis; 2 to chronic pancreatitis.

The diagnosis of pancreatic collections is discussed from a clinical point of view but little of a specific nature is to be found until the palpating hand locates a mass in the epigastrium; only then can we be suspicious. Serum amylase determination is of great value and roentgenographic studies were found of use. The routine examinations showed a mass in the general region and gastric pneumograms [see paper by Thomas, on page 128 of this issue of RADIOLOGY] aided further in the localization. However, the diagnosis cannot be made preoperatively with certainty.

THE PERITONEUM

Diffusion and Localization of Experimental Infections of the Peritoneum. Bernhard Steinberg and Ruth A. Martin. Surg., Gynec. & Obst. **79:** 457–468, November 1944.

A previous contribution by Steinberg (J. A. M. A. **116:** 572, 1941) showed the diagnostic and prognostic value of study of the exudate drawn from the midperitoneal cavity. The present paper endeavors to refute the prevailing view that peritoneal infection spreads from a focus against a defense barrier set up by the body and that a diffuse infection of the peritoneal cavity is inimical to recovery. The authors contend

that the infection is at once disseminated fairly uniformly in order that the body defenses can better deal with it.

Experimental infections of the peritoneal cavity were induced in dogs by various methods—ligation of the appendix and administration of castor oil; injection of air and cultures of bacteria into the lumen of the appendix; injection of infectious material into the pelvic cavity. Spread of the infection was followed by injecting iodized oil along with the infectious material and following its dissemination by x-ray studies. Two dogs were killed at different intervals and the peritoneal cavities investigated.

It was found that diffusion throughout the peritoneal cavity after appendiceal perforation was rapid (twenty minutes to two hours). The spread was surprisingly uniform, and the exudate showed a uniform bacterial count throughout the peritoneal cavity except on the surface of the appendix and in the right subdiaphragmatic area, where the count was much higher. Localized accumulations (abscesses) occurred in animals showing signs of recovery.

The advantages of diffusion of the bacteria over a large peritoneal surface are threefold: (1) The microorganisms are distributed in smaller numbers, so that there is a more efficient and speedier phagocytosis. (2) A greater number of phagocytic leukocytes are able to migrate into the peritoneal cavity and probably act at an accelerated tempo. (3) Passage of a large number of bacteria through the lymphatic and capillary vessels is made possible.

Conditions which may bring on a fatal outcome are: (1) the presence in the peritoneal cavity of environmental conditions which delay bacterial removal or increase the rapidity of bacterial multiplication; (2) an extensive initial or a continuous outpouring of bacteria.

The observation that bacteria and leukocytes accumulate in the right subdiaphragmatic region in greater numbers than elsewhere in the peritoneal cavity is interesting from the point of view of formation of a subphrenic abscess. Changes in posture did not appear to alter appreciably such tendencies. The suction action of the diaphragm and the anatomic passageway formed by the right lateral gutter may account for the greater accumulation of bacteria and leukocytes in that region. Massive bacterial invasion of the area with a resultant vascular stasis and closure by inflammation of capillary and lymphatic vessels by which microorganisms are removed probably completes the process of subphrenic abscess formation.

In the light of their findings the authors question the wisdom of efforts to delimit and circumscribe the infectious process. They suggest the terms, "regressing and progressing" peritonitis to indicate, respectively, favorable and unfavorable progress, and would discard the current designation of "diffuse" or "spreading" peritonitis. J. L. BOYER, M.D.

THE SPLEEN

Solitary Calcified Cyst of the Spleen. Report of a Case. H. W. Neidhardt. J. Kansas M. Soc. **45:** 382–383, November 1944.

A 56-year-old male was struck about the region of the lower left chest when the ditch in which he was working caved in. Pain on deep breathing followed, with considerable tenderness in the left upper quadrant and lower chest and limitation of respiratory movements. Roentgenograms revealed no fractures, but a nearly circular, sharply outlined mass was seen in the left upper quadrant. The left side of the diaphragm was elevated, the apex of the heart was displaced considerably to the left, and the transverse colon and left kidney were displaced downward on the left side. Two weeks later a laparotomy was performed and the mass was identified as a calcified cyst in the splenic region. At its upper pole numerous fairly recent adhesions were present. The cyst, which was ruptured in its removal, contained approximately 1,000 c.c. of thick chocolate-colored fluid, with a large number of minute shimmering crystals. Since the spleen was not found, it was assumed to be incorporated in the cyst. The pathologic diagnosis was solitary calcifying cyst of the spleen, acute and chronic splenitis, and perisplenitis. The patient made an uneventful recovery following the splenectomy.

THE SKELETAL SYSTEM

Severe Osteitis Fibrosa Cystica with Parathyroid Tumor. Report of a Case of Fifteen Years' Duration. Donald E. Coburn. Am. J. Surg. **66:** 252–258, November 1944.

A severe case of generalized osteitis fibrosa cystica due to a parathyroid adenoma located in the superior mediastinum is reported.

A 60-year-old woman complained of severe pain in the right hip radiating down the posterior aspect of the right thigh, of six months' duration. During this time she had become progressively shorter, had worn a brace to support her back, and required the aid of crutches. In the previous 16 years she had suffered no less than four fractures and had undergone one operation for kidney stones.

The patient's features were coarse and puffy, the skin was dry and subicteric, and the hair dry and brittle. The thyroid was not enlarged, but there was a questionable fullness opposite the lower pole on the right. The entire left side of the chest was compressed and flattened toward the right, producing a modified pigeon-breast deformity. The right side of the chest flared, rib spaces were prominent, and respiratory excursion on this side was nearly normal. The spine was more or less rigid, with a prominent dorsal kyphosis and an accompanying right roterolateral dorsal scoliosis. Motion in the right hip was painful and restricted in all directions. Reflexes were sluggish; cutaneous sensation was normal.

X-ray examination of the skull showed the calvarium to be thickened, while the bones of the vault had a fine, mottled, granular appearance. The maxillary and sphenoidal sinuses appeared enlarged. Roentgenograms of the upper extremities showed areas of absorption with cyst formation in the upper ends of both humeri, the left ulna, the right radius, the right os magnum, the third left metacarpal, and in the proximal phalanx of the right first and the left second fingers. The site of a previous fracture of the right humerus could be easily seen. In the right femur a mass of thin-walled cystic cavities completely replaced the normal bone structure of the upper two-thirds. The overlying cortex was extremely thin and somewhat expanded. Cysts were also present in the right tibia, the left tibia, both patellas, and in the first left meta-

tarsal. A film of the abdomen showed numerous areas of increased density in both kidney regions, consistent with the diagnosis of nephrocalcinosis. Films of the pelvis revealed extensive involvement of both innominate bones with compression and distortion of the entire left half of the pelvis upward and inward to create a typical oblique deformity. There were large cystic areas of bone destruction in the region of the right acetabulum and right ilium. Multiple areas of cystic formation were present in the ribs, and there was almost complete loss of substance of the third, fourth, fifth, and sixth dorsal vertebrae.

The patient's history and the x-ray findings suggested a diagnosis of osteitis fibrosa cystica due to parathyroid adenoma. An elevated blood calcium with a decreased blood phosphorus and increased blood phosphatase confirmed the diagnosis.

At operation, a tumor measuring approximately 3.5 × 2 cm. was found in the superior mediastinum. This was removed without difficulty. Convalescence was marked by a fracture of the right femur. Films taken about ten weeks following operation, however, showed the fractured femur to be in good alignment, with some callus formation. There was also evidence of beginning recalcification throughout the entire skeletal system. About four months later continued recalcification was demonstrable; many of the cystic areas in the extremities had begun to fill in and the surrounding cortex was appreciably increased. Films of the abdomen at this time showed the areas of nephrocalcinosis in both kidney regions to be essentially the same as on previous examinations. For this reason the prognosis must be guarded, as it has been shown that such deposits may ultimately result in death through the continued destruction of kidney tissue long after the primary tumor has been removed. Numerous roentgenograms are reproduced.

Importance of Degeneration of the Cervical Disks for the Narrowing of the Intervertebral Foramina. Fred Haglund. Acta radiol. **23:** 568–580, Dec. 15, 1942.

An account is given of the result of an anatomical examination of degeneration and protrusion of the cervical disks. Degeneration of the disks is often followed by protrusion, which may occur posteriorly, in the direction of the spinal canal; postero-laterally, in the direction of the entrance of the intervertebral foramina; laterally, in the direction of the external part of the intervertebral foramina and vertebral artery. The most important observation, with regard to the occurrence of brachialgia, concerns the conditions following disk protrusion in a postero-lateral direction. The author has discovered and describes cases of narrowing of the intervertebral foramina caused by disk protrusion with ensuing pressure on the spinal nerves.

MAX SGALITZER, M.D.

Dilatation of the Vertebral Canal Associated with Congenital Anomalies of the Spinal Cord. A. Earl Walker. Am. J. Roentgenol. **52:** 571–582, December 1944.

Four cases are presented in detail to illustrate the fact that dilatation of the spinal canal may occur as the result of congenital anomalies of the cord structures. In 3 of the cases the enlargement of the canal was associated with myelodysplasia. In the fourth there was a vascular anomaly of the cord. Except in this last case, there was no evidence of pressure on the spinal cord so that the enlargement of the canal presumably occurred early in the course of development, probably at the time the ossification centers were laid down. In one of the cases the enlargement occurred in the lumbosacral region and was associated with myelodysplasia and a diffuse lipoma infiltrating the roots of the cauda equina. In the second case, also in the lumbar region, widening of the interpedicular spaces was associated with diplomyelia, the cord being split into two components, and other anomalies. In the third case a similar splitting of the cord was found in the cervico-dorsal region, again associated with widening of the interpedicular spaces at this level.

Although neoplastic conditions cause most instances of roentgenographically demonstrable enlargement of the spinal canal, these cases emphasize that congenital abnormalities of the spinal cord must also be considered as etiologic factors. L. W. PAUL, M.D.

Role of the Nucleus Pulposus in the Pathogenesis of So-Called Recoil Injuries of the Spinal Cord. Fritz Cramer and Francis J. McGowan. Surg., Gynec. & Obst. **79:** 516–521, November 1944.

In the condition known as "recoil" injury, the spinal cord may be severely damaged without demonstrable or commensurate injury to the spinal column, the usual causes being diving or automobile accidents. Forceful anterior flexion, the head being "whip-lashed" forward, is the mechanical cause. The pathogenesis of this injury has been believed to be sudden massive dislocation of the vertebral body, which then recoils by reflex contraction of the antagonist muscles. The authors, however, question this explanation, their theory being that the injury to the cord is due to the violent protrusion of the intervertebral disk or to rupture of the nucleus pulposus when subjected to sudden intense compressive force.

The report of a case of injury due to diving into shallow water is presented. A traumatic paralysis, a quadriplegia, developed, and death occurred fifty hours after the accident. A lateral film of the cervical spine showed a slight compression fracture of the anterior portion of the fifth cervical vertebra as well as a slight narrowing of the posterior portion of the intervertebral space, without any evidence of dislocation of the vertebral body. Autopsy confirmed a simple fracture of the body and arch of the fifth cervical vertebra plus acute traumatic prolapse of the intervertebral disk between the fourth and fifth cervical vertebrae. Opposite the fifth intervertebral space, the spinal cord was practically severed except for the posterior columns and the posterior portion of the pia. The case illustrates laceration of the spinal cord by sudden, severe traumatic prolapse of the intervertebral disk by the projectile force of the compressed nucleus pulposus.

The authors found, in reviewing the literature, that there were descriptions and photographs of somewhat similar cases, showing herniations of the nucleus pulposus; yet the cord injury was usually attributed to dislocation with recoil. This momentary extreme dislocation with immediate spontaneous reduction is held to be improbable on anatomic grounds. The authors believe that the autopsy findings in other cases, as well as their own, point to traumatization of the cord by the intervertebral disk as a result of compression of the nucleus pulposus or its herniation.

ROBERT E. BOOTH, M.D.

Painful Shoulder Due to Lesions of the Cervical Spine. Bernard N. E. Cohn. Am. J. Surg. **66:** 269–274, November 1944.

This paper deals with lesions of the cervical spine which may cause a painful shoulder with radiation of pain into the arm. When a patient presents himself for the relief of pain in the shoulder region and arm, a large variety of conditions must be considered, including hypertrophic arthritis of the cervical spine, herniated intervertebral disk, cervical rib, the various neuritides, and local lesions of the shoulder region, as arthritis, periarthritis, bursitis, and tears of the supraspinatus tendon. Diagnosis can be made only after careful and detailed physical and roentgenographic examination. The cervical spine should be examined routinely in all cases of painful shoulder in which there are insufficient signs to verify the diagnosis of a local lesion or neuritis and in those cases of presumptive shoulder pain which do not respond to therapy within a reasonable period of time.

Four cases (hypertrophic arthritis of the cervical spine; metastatic carcinoma of the cervical spine; tuberculosis of cervical spine; herniated cervical intervertebral disk) are presented to show the difficulties involved in arriving at a correct diagnosis.

Etiology of Phosphorus Sclerosis. Olov Fr. Holm. Acta radiol. **23:** 549–561, Dec. 15, 1942. (In German.)

Since the enactment (1929) of an insurance law for occupational diseases in Sweden, one case of phosphorus poisoning has occurred in which payment had to be made. Phosphorus sclerosis originates during processes of ossification. In severe cases hard zones are produced which may last during the life of the patient; in milder cases the sclerotic zones may disappear completely.

Three stages of chronic phosphorus poisoning are described: (1) the stage of calcium deposition, represented by broad and dense epiphyseal lines (the changes at this stage are reversible, and if the exposure is discontinued, the lines may entirely disappear); (2) the stage of persistence of epiphyseal lines, in which deposition and absorption counterbalance each other; (3) the stage of bony atrophy, osseous fragility, impairment of fracture healing, and tendency to bone necrosis (especially in the jaw). Bone necrosis itself is due to infection in atrophic bone damaged by the effect of phosphorus.

The histories of 4 patients, 3 of whom had worked in match factories, are recorded, and roentgenograms are reproduced. MAX SGALITZER, M.D.

Aseptic Bone Necrosis of the Os Capitatum (Os Magnum). Gunnar Jönsson. Acta radiol. **23:** 562–564, Dec. 15, 1942. (In English.)

The author describes a case of aseptic necrosis in the os capitatum in a 22-year-old female with no history of trauma. The picture resembled, both clinically and roentgenologically, the changes characteristic of malacia of the os lunatum (Kienböck's disease).

MAX SGALITZER, M.D.

March Fractures, a Series of Fifty-Eight. Walter A. Dew and James H. Wooten, Jr. Mil. Surgeon **95:** 356–359, November 1944.

Fifty-eight cases of fracture of the metatarsus in 55 trainees are reported. Fifty-three men successfully completed the course of training in a replacement center with physical activities which included marches up to 25 miles. Two patients had subsequent march fractures, the first, one, and the second, two.

Roentgenograms of the foot taken immediately after onset of symptoms may not reveal the fracture line. Treatment is begun at once in all patients with signs of march fracture, and the x-ray examination is repeated in one week in those with initial negative roentgen findings. At this time evidence of callus formation will reveal the site of the fracture even though the fracture line is still not discernible. This follow-up roentgenogram proved to be essential.

The authors advise that patients with march fractures be treated as outpatients, with the Army shoe as a splint whenever possible. Prophylactic measures—gradual increase in the length of marches and in the weight of equipment and marching on soft rather than hard ground—were found to reduce the incidence of these fractures.

GYNECOLOGY AND OBSTETRICS

Roentgenologic Diagnosis of Placenta Previa. G. Sohrne. Acta radiol. **23:** 541–548, Dec. 15, 1942. (In English.)

A method is described by the author by means of which the presence or absence of placenta praevia may be demonstrated. Air is introduced as a contrast medium into the urinary bladder and the rectum. The catheter is then withdrawn. Frontal roentgenograms are supplemented by views taken in oblique projections in both directions, and, when necessary, in the lateral position.

In some cases, the placenta is directly visualized. In most cases, conclusions are based on the size and configuration of the interspace between the upper pole of the bladder and the fetal head.

MAX SGALITZER, M.D.

THE GENITO-URINARY TRACT

Perirenal Insufflation. Fedor L. Senger and John J. Bottone. Am. J. Surg. **66:** 213–219, November 1944.

In cases in which overlying malpositioned organs, pocketed intestinal gas and contents, or perirenal adhesions and perinephritis obscure the kidney borders, the authors have found perirenal air injections of value. By surrounding the kidney with an envelope of air, they have obtained useful added information in unusual cases of ptosis, rotations or failure of rotations, malformation, and agenesis or atrophy of the kidney. The procedure also supplies knowledge concerning the local spread and operability of renal tumors. Complications are infrequent and of minor degree if ordinary care is taken. A series of 175 cases of perirenal air injection without serious mishap is presented. The technic is described. Roentgenograms are reproduced.

Reflex Anuria Following Retrograde and Excretory Investigation. Benjamin Levant and Krikor Yardumian. Urol. & Cutan. Rev. **48:** 554–556, November 1944.

So-called reflex anuria may be due to a definite pathological factor. In other instances, no obvious lesion is present, and it is with such cases, particularly follow-

ing pyelography, that the authors are concerned. Anuria of this type may be due to shock or inhibitory reflex. The blood secretory pressure drops and secretion of urine fails to take place. In another group of cases anuria may be due to a disturbance of intratubular pressure. In retrograde pyelography, the renal pelvis and calices are distended under pressure and when this is released the intratubular pressure is altered, causing congestion and edema of the remaining kidney tissue with compression of the capillary beds. Still other cases are attributed to inadequate elimination of the radiopaque substance, which may be precipitated in the tubules, causing urinary retention.

Two cases of anuria are reported. One followed cystoscopy and is attributed to a psychic factor. The other was incident to intravenous urography with diodrast following cystoscopy. The authors ask: "Is anuria another type of reaction to diodrast or is it that the cystoscopic manipulation produced inhibitory or shock type of prerenal anuria?" They conclude: "It is probable that the urethro-renal reflex is only depressant and the final blow is given by the intravenous injection of diodrast while the renal tissue is not up to par." They believe with Quinby and Austen (New England J. Med. **221:** 814, 1939. Abst. in Radiology **35:** 259, 1940) that the intravenous study should be delayed several days in these cases.

MAURICE D. SACHS, M.D.

Method of Locating a Mobile Renal Calculus at Operation. B. W. Goldstone. Brit. M. J. **2:** 77–78, July 15, 1944.

The author points out that kidney stones have considerable freedom of movement within the bounds of the kidney and are therefore difficult to locate and remove surgically.

The splitting operation is bad on account of resulting hemorrhage; palpation is rarely successful in case of small stones; probing with a single needle is unreliable, and simple roentgenograms are frequently of doubtful value in localization because a short pedicle commonly exists. This factor makes for considerable distortion in the film.

In the method advised, a row of needles is inserted longitudinally converging to the center of the pelvis. An x-ray film localizes the stone in one plane with relation to one of these needles. After the others are withdrawn, a second row is inserted at right angles to the first. Another film is made and the stone is now localized in two planes and in close proximity to one pin. It is then a simple matter to dissect around the remaining pin and find the stone. Q. B. CORAY, M.D.

Patent Urachus. Ralph W. Nichols and Robert M. Lowman. Am. J. Roentgenol. **52:** 615–619, December 1944.

A case of patent urachus in which roentgenographic visualization was employed for diagnosis is reported. A newborn female infant had a protruding cylindrical mass at the umbilicus, the center of which contained mucosa; from it a small quantity of fluid resembling urine was expressed. Injection of diodrast into the opening demonstrated a urachal stalk communicating freely with the bladder. Subsequent urographic studies with the aid of subcutaneously injected diodrast confirmed this finding.

Anomalies of the urachus are of four types: (1) The urachus is patent throughout, the complete type. (2) It is patent at its umbilical end only, the blind external type. (3) It is patent at the vesical end, the blind internal type. (4) Both ends are closed, but there is failure of closure of some part between the ends; this type gives rise to the urachal cyst.

The present case is of the first type. All types of urachal anomaly appear to be rare. Thus Herbst, in 1937 (South. M. J. **30:** 711, 1937) was able to find only 148 cases recorded in the medical literature of the past four centuries. L. W. PAUL, M.D.

RADIOTHERAPY

NEOPLASMS

Malignant Goiter. Robertson Ward. Surgery **16:** 783–803, November 1944.

Since malignant change is of exceptional occurrence in diffuse toxic goiter, the problem of cancer of the thyroid gland may be said to revolve about nodular goiter. Primary carcinoma arising from a previously normal gland, however, is probably less rare than statistics would suggest. The mere fact that a tumor has existed for many years previous to its surgical removal does not necessarily indicate that the growth was benign at its inception.

Two-thirds of the patients with carcinoma of the thyroid are seen between the ages of forty and seventy years, but it should be borne in mind that aberrant thyroid tissue undergoes malignant change at a much earlier age.

As would be expected, malignant tumors of the thyroid occur in greater number in endemic goiter regions. The incidence of malignant change in surgically treated goiters, as given by various authorities, ranges from less than 1 to 6.5 per cent. While the actual number of men with malignant goiter is less than the number of women, the expectancy of carcinoma in men with nodular goiter is much greater.

All the diagnostic signs of malignancy are based upon penetration of the capsule and involvement of adjacent tissues. Hoarseness results from infiltration of the recurrent nerves; fixation is caused by infiltration of the trachea or ribbon muscles; hardness is due to outward growth of cancerous tissue. All of these three cardinal signs can be found, however, in benign nodular goiter.

From a prognostic and therapeutic standpoint, thyroid cancers can be roughly arranged in three groups: Group I, papillary carcinomas; Group II, malignant adenomas; Group III, all others, including scirrhous, small-cell, and large-cell types, undifferentiated carcinomas, and those with a sarcomatous tendency.

Prognosis is based on the time of diagnosis, the pathologic pattern, and the presence of metastases. The easier the diagnosis the worse the prognosis. In cases diagnosed preoperatively as cancer, or suspected cancer, the five-year-survival rate is 20 per cent; in those in which cancer is discovered at operation, 40 per cent; in those in which diagnosis is first made upon section of the specimen, 80 per cent. Papillary carcinomas have the most favorable outlook. Indeed, the degree of

malignancy of a given tumor can apparently be estimated by the extent of its departure from that pattern.

Treatment is prophylactic, definitive, and palliative. Enucleation of adenomas is a relatively simple procedure and is highly effective as a prophylactic measure. When a report of carcinoma is unexpectedly returned, reoperation to remove the involved lobe and suspected soft tissue is justified. The operation should be extensive enough to remove all involved structures and yet preserve at least one recurrent nerve, one parathyroid gland, and both carotid arteries. Tumor invading the trachea should be excised flush with the tracheal wall. Mediastinal extension of malignant tissue often proves to be the unsurmountable obstacle to complete removal of the tumor but several procedures have been devised to accomplish this result.

Roentgen therapy can be used alone for palliation or combined with surgery in an attempt to eradicate the disease. Even in advanced cases, a biopsy should be performed before irradiation is carried out. This offers the advantage of establishing the diagnosis and preventing irradiation of a pure papillary tumor, which may break down and ulcerate because of its extreme radiosensitivity.

The principal value of x-ray therapy lies in its administration postoperatively, for the elimination of cancerous tissue not detected or not removable at operation. Such tissue is frequently located in the mediastinum, and for this reason the mediastinal space should be included in the area treated. Three thousand roentgens delivered to the tumor bed are considered to be the lower limit of effective dosage. The best results can be expected to occur in tumors shown to be predominantly papillary. Except in these papillary tumors and in a small number of malignant adenomas, irradiation seems to be of little value. No case of anaplastic large- or small-cell carcinoma, or carcinosarcoma has shown a favorable response to treatment either by surgery or irradiation.

The results of treatment of locally recurrent tumors and metastatic deposits are influenced even more strongly by the pathologic pattern of the growth than are the results of treatment of primary neoplasms. If the metastases are from a well differentiated tumor predominantly papilliferous, their response to irradiation is usually good. Locally recurrent nodules, especially if the primary growth was a malignant adenoma, respond poorly to irradiation and should be removed.

J. E. WHITELEATHER, M.D.

Synovial Sarcoma. Cushman D. Haagensen and Arthur Purdy Stout. Ann. Surg. **120:** 826–842, December 1944.

A considerable number of cases have been reported as synovial sarcomas which in reality were xanthomatous giant-cell tumors, tumor-like hyperplasias of the synovial tissues, or other benign lesions curable by simple excision. The apparent cure rate for synovial sarcoma has thus been made to appear higher than it actually is.

To obtain confirmation of the mesothelial nature of the neoplasm, tissue culture studies were carried out in 3 cases. This work is reported by Murray, Stout, and Pogogeff (Ann. Surg. **120:** 843, 1944). The three tumors from which cultures were obtained had striking histologic characteristics. They were composed of an inextricable admixture of mesothelial cells, which often lined slits or tubes and secreted a mucicarminophilic substance, with strands of active, hyperchromatic fibrosarcoma-like cells associated with reticulin fibers. The relative amounts of these two cell forms varied enormously, but both were always present.

With these characteristic histologic features in mind, cases reported as synovioma, synovial sarcoma, or designated by some related term, were re-examined and those which did not fulfill the criteria were rejected. This screening yielded 95 cases, to which the authors add 9 previously unreported examples.

The condition occurs preponderantly in males, the proportion being 3:2. Although the tumor may develop at any age, it is more frequent in early adult life. The mean age of the 103 patients whose age is known was 32 years. Nearly one-half of the tumors developed in the region of the knee joint.

Of the 104 patients with synovial sarcoma, only 3 are known to be free from evidence of persistence or metastasis more than five years after treatment. The therapy employed has been of four general types: (1) radiation alone or in combination with surgery; (2) local excision, often repeated several times; (3) excision, or exploratory operation with an attempt at excision, followed immediately, or within a short time, by amputation; and (4) biopsy followed shortly by amputation.

Radiation appears to have been singularly futile in this disease. It has often been employed, but five-year cure has not been obtained in a single case in which radiation was the chief reliance. One patient was well six years after treatment, but in this instance irradiation was followed by amputation. The authors were unable to find any clear evidence that radiation is even of palliative value.

Local excision was undertaken in 84 of the 104 cases tabulated. As with other forms of fully malignant sarcoma, any sort of local excision almost always fails to cure. Secondary amputation for recurrence following local excision was performed in 26 of this group of conservatively treated patients, but none was cured.

Excision, or exploratory dissection with an attempt at excision, followed immediately or within a short time by amputation, was carried out in 7 patients. Only one of these patients appears to have been cured.

In 4 cases, a simple biopsy was done, followed shortly by primary amputation when the malignant nature of the lesion became clear from histologic study. Although 3 of these patients subsequently had pulmonary metastases, the fourth has been well for eight years. This is the longest survival on record following any form of treatment for synovial sarcoma. This case, one of the authors', is reported in detail.

The authors believe that the most radical surgical attack—carefully limited biopsy and a high immediate amputation—is the rational therapy for synovial sarcoma. A biopsy must be done because it is impossible to diagnose synovial sarcoma, as well as a variety of other deep soft-part tumors, from the clinical picture alone. An incisional biopsy is essential, for the diagnosis of the tumor rests to a considerable extent upon the histologic architecture. The dissection should be as limited as possible. The diagnosis having been proved histologically, amputation should be performed promptly and at high enough level to avoid local persistence of the disease. Since regional lymph node dissection is not a hazardous operation, it should be seriously considered as a separate and final stage in the treatment of synovial sarcoma.

The Place of Surgery in Fibroids of the Uterus. Channing W. Barrett. Am. J. Surg. **66:** 148–156, November 1944.

This is a more or less general discussion of surgical and radiation therapy of fibroids of the uterus, written from the point of view of the surgeon. The author believes that radiation should be used only in small tumors occurring at the menopause, presenting no complications and no symptoms beyond slight or moderate bleeding.

Some Effects of Testosterone, Testosterone Propionate, Methyl Testosterone, Stilbestrol, and X-Ray Therapy in a Patient with Cushing's Syndrome. Martin L. Deakins, Harry B. Friedgood, and Joseph W. Ferrebee. J. Clin. Endocrinol. **4:** 376–384, August 1944.

The metabolic and clinical effects of testosterone propionate, stilbestrol, methyl testosterone, and testosterone were observed under controlled conditions in a fifteen-year-old girl with Cushing's syndrome. Injections of testosterone propionate, 25 mg. per day, caused a diminution in nitrogen excretion and a disappearance of creatinuria. Stilbestrol, 2 to 10 mg. per day, intramuscularly, did not affect nitrogen excretion but perhaps induced a moderate increase in creatine excretion. Methyl testosterone, 40 mg. per day, orally, caused a striking decrease in nitrogen excretion and a tremendous increase in creatine excretion. Creatinine excretion remained unaltered. Testosterone, 40 mg. per day, orally, did not produce unequivocal changes in excretion of nitrogen, creatine, or creatinine.

Irregular increases in 17-ketosteroid excretion, with levels as high as 26 mg. per day, occurred during administration of testosterone propionate. During recovery from the effects of this hormone and also in the period of stilbestrol administration, the 17-ketosteroid excretion averaged about 12 mg. per day. Methyl testosterone caused an initial 3-day increase in 17-ketosteroid from 15 up to 25 mg., followed by a depression to levels of about 5 mg. Free testosterone caused a precipitous and marked rise in 17-ketosteroid excretion. Thirty-eight to 48 mg. of ketosteroid was excreted on the 40-mg. dose and over 100 mg. on the 150-mg. dose. The pattern of biochemical activity of these hormones suggests marked differences in their metabolism.

The androgens induced distressing accentuation of acne, hirsutism, and hypertrophy of the clitoris. Stilbestrol improved these conditions, but the benefit was not striking and was associated with certain untoward reactions and a transient hyperglycemia.

Three series of roentgen irradiation were given, each consisting of eight daily exposures of the hypophyseal region through two portals (right and left temporal). A total of 6,480 r was administered in a period of ten months. This therapy appeared to improve the subjective and objective manifestations of the disease.

NON-NEOPLASTIC DISEASES

Effect of Grenz Rays on Leprous Infiltrations: Report of an Attempt to Influence Leprous Infiltrations by Roentgen Rays of Long Wavelength. Felix Sagher. Arch. Dermat. & Syph. **50:** 311–314, November 1944.

There is no specific therapy for leprosy. In fact, no therapeutic measure now known can even influence a localized lesion.

Radium and x-ray have been tried on leprous nodules and, while there have been some favorable results, severe damage has occurred to the surrounding skin and underlying tissues. As grenz rays do not penetrate deeply and their effect on underlying tissues would therefore be negligible, the author decided to try their effect in two patients with relatively superficial lesions which had failed to respond to the usual methods of treatment. Each of the patients was treated in nine different areas, the lesions being of varying size and thickness. The treatment factors were 6 to 14 kv., equivalent of half-value layers of 0.021 to 0.033 mm. aluminum, 10 ma., 10 cm. focal skin distance. The total dosages were from 600 to 8,000 r to an area, given in four to six weeks. On one area a single dose of 5,000 r was given at one time. The results have been followed over periods of six months to two years and nine months.

If more than 2,000 r was given to an area, the lesion would disappear in an interval of between four months and a year. There was some atrophy of the skin, but this was not greater than in a similar area where a lesion had disappeared spontaneously. There was severe atrophy of the skin in the area that was treated with 5,000 r at one time. Even though the lesions disappeared, Hansen bacilli were constantly found in the skin at each test.

In both patients new lesions developed under observation. "It was a particularly striking feature that newly developing infiltrations in the surrounding skin halted at the border of the treated areas."

JOSEPH T. DANZER, M.D.

Use of Radon to Prevent Otitis Media Due to Hyperplasia of Lymphoid Tissue and Barotrauma (Aero-Otitis). Edmund P. Fowler, Jr. Arch. Otolaryng. **40:** 402–405, November 1944.

In England, American soldiers have been found more prone to have hyperplasia of pharyngeal lymphoid tissue, secondary eustachian salpingitis, and consequent otitis media than in the United States. In the ear, nose, and throat section of one general hospital, 0.8 per cent of ground personnel and 22 per cent of flying personnel had a history of recent otitis when examined. Nearly all of these patients had sufficient excess lymphoid tissue around the eustachian tubes to account for this. From December 1942 to March 1944, 220 patients were treated with radon for this condition. Capsules made of platinum 0.65 mm. thick, measuring 3.5 mm. × 15 mm., attached to a semimalleable 18 gauge wire, 14 cm. in length, were used. Each capsule contained 75 millicuries of radon. Two radon capsules (totaling 150 mc. the first day) are passed along the floor of the nose and then separated by placing a small piece of rubber tubing over the insertion wires outside the nostril. They are left in place twenty-six minutes the first day, giving a dosage of 66 milligram hours. The radon capsules as a rule lie very near any exuberant lymphoid tissue on the medial side of the torus tubarius. For convenience they are considered as a single source in estimating dosage. Since the capsules are separated, however, the total effect of 66 milligram hours does not reach any area in the nasopharynx. Originally the treatments were given at intervals of six weeks until four to six treatments had been administered. Later the interval between treatments was reduced to three weeks, with no untoward results. [The author reports, in a footnote, that since the submission of this article

he has found 100 milligram hours with filtration of 0.65 mm. of platinum at four-week intervals much more satisfactory than the use of 66 milligram hours at three-week intervals. With this method, often two treatments have been sufficient, although three were usually necessary and four were given to maintain the improvement.]

Airmen with subacute blockage of the eustachian tube often get along perfectly well on the ground or even at low altitudes, but when they fly high or are subjected to rapid changes of altitude, the eustachian tubes close so that they cannot be opened. This otitic barotrauma produces conditions varying from a mild catarrh of the middle ear to acute, severe otitis with or without rupture, and the involvement may even go on to an acute purulent process. None of these conditions develops if the eustachian tubes are functioning properly. Most of the malfunction is due to swollen tissue within them. In addition to lymphoid tissue, there are frequently swollen glands, edematous mucosa or submucosa, and excessive secretion blocking the tubes. The cold air of high altitudes often causes hypersecretion in the nose, and the secretion bathes the tubal orifices and blocks or irritates them. Apparently radiation reduces the activity of the glands, shrinks the edematous tissue, and has a specific effect on hyperplastic lymphoid tissue. It is probable that the radiation also has a beneficial effect on the infection present.

American soldiers after a few months in England often have so much excess lymphoid tissue that as soon as the mouth is opened this tissue can be seen bulging from the posterior pharyngeal wall or behind the posterior tonsillar pillars in heavy bands. It is particularly prominent following virus bronchopneumonia. Radon rays in the dosage given do not sufficiently reduce large masses of hyperplastic lymphoid tissue in the midline, and 24 patients in this series underwent adenoidectomy.

Of 29 airmen with recurrent otitis media treated with radon (200 mg. hr. or more) on whom the follow-up investigation is complete, 23 are flying again and 28 are free of otitis. Seventy-five per cent of the ground personnel who had sufficient treatment were free of otitis media for at least four months. Some of this group probably would have become well without treatment and eventually some of them will have recurrences. The author believes that treatment of the lymphoid tissue about the eustachian tube with radon or radium is infinitely more efficacious and the effects more permanent than those of catheterization, politzerization or bougienage. It requires, however, the continual guidance of a qualified radiologist.

RADIATION EFFECTS

Clinical and Roentgen Aspects of Irradiation Stricture of the Rectum and Sigmoid: Its Course and Treatment. H. C. McIntosh and J. E. Hutton. Am. J. Roentgenol. **52:** 647–662, December 1944.

Upon the basis of a series of 44 cases of post-irradiation injury of the bowel, the authors discuss the types of damage which may occur and their medical management.

The primary damage in cases of gross bowel injury after pelvic irradiation appears to be to the smaller hemorrhoidal vessels supplying the bowel, with thrombotic occlusion resulting in infarction and mucosal ulceration. The principal clinical complaint is diarrhea, which may occur as early as the third week after the beginning of treatment or not until some months later. The late development of symptoms may suggest an extension of the original lesion to involve the bowel, but this is uncommon in the authors' experience. Diagnosis is made by a carefully taken history, roentgen studies, sigmoidoscopy, and biopsy whenever possible.

The condition is best treated conservatively. Bed rest and a bland diet are indicated during the acute stage. Antispasmodics are helpful. A 6-oz. retention enema of 1 per cent tannic acid helps to control bleeding. If signs and symptoms of obstruction supervene, surgical treatment may be necessary.

Severe and extensive post-irradiation damage of the bowel often undergoes healing with very little functional impairment, though marked anatomic defects may be present. Such an outcome is materially assisted by careful medical treatment. L. W. PAUL, M.D.

Dwarfism Associated with Microcephalic Idiocy and Renal Rickets. S. J. Glass. J. Clin. Endocrinol. **4:** 47–53, February 1944.

The author records a case of bizarre dwarfism associated with microcephalic idiocy and renal rickets, in a girl who died at the age of thirteen, of an intercurrent infection. When the patient's mother became pregnant she was suffering from inactive pulmonary tuberculosis and active tuberculous osteomyelitis of the sacro-iliac joint with a draining sinus. Because of preceding amenorrhea of about nine years' duration she was unaware of the pregnancy until about the third month. She then requested a therapeutic abortion. This was attempted by roentgen irradiation of the pelvis, without success. The irradiation, however, was persistently carried on at weekly intervals from the third to the eighth month. The dosage of roentgen radiation sustained by the growing fetus is not known. The pregnancy was finally terminated in the eighth month by cesarean section. The infant weighed two and three-quarters pounds. Dwarfism and imbecility became apparent in infancy, but there was no roentgen evidence of rickets until six months before the patient's death, and no obvious rickets was ever observed clinically. At autopsy extreme nervous and somatic hypoplasia was seen. This is considered to have been the result of brain damage induced by the roentgen irradiation and renal rickets.

Injury of Bones by Roentgen Treatment of Cancer of the Uterine Cervix. Frithiof Truelsen. Acta radiol. **23:** 581–591, Dec. 15, 1942. (In English.)

The author reports three cases of spontaneous fracture of the femoral neck following intensive x-ray treatment for cancer of the cervix. In one case a microscopic examination of the bone tissue was made. Destruction of the bone marrow was found with features resembling an osteitis fibrosa and with necrosis of the bone tissue. There was complete lack of osteoblasts and osteoclasts.

The author believes that the influence of the x-rays on osteoblasts and osteoclasts is probably the chief cause of osseous changes following irradiation. Vascular changes followed by nutritive disturbances may also play an important part.

Since osseous changes in the femur may complicate radiotherapy of uterine cancer, radiographic examination of the pelvis is indicated in all cases of hip pain and

gait disturbances in order to exclude fractures, coxa vara, and halisteresis. MAX SGALITZER, M.D.

Mathematical Theory of Biologic Reaction Phenomena, Especially Following X-Ray Irradiation J. Th. van der Werff. Acta radiol. **23:** 603–621, Dec. 15, 1942. (In German.)

In the course of a critical consideration of the mathematical theory of R. M. Sievert concerning reaction phenomena following irradiation, the author expounds a new mathematical theory, for details of which the reader must consult the original paper. According to his theory, the reactions after roentgen irradiation are subordinated to the general biological reactions. Laying the ground work for the theory by some simple differential equations, he proceeds by solving these equations and by discovering a number of formulae and properties from the solutions. With the help of this theory, a great number of experimental facts can be explained. Among such facts are the findings of Arne Forssberg with regard to the rate of growth of Phycomyces blakesleeanus following x-ray irradiation.

MAX SGALITZER, M.D.

Radon/Carbon Dioxide Ratio as an Index to Excessive Radium Absorption. Frank E. Hoecker. J. Indust. Hyg. & Toxicol. **26:** 281–288, November 1944.

It has become the accepted practice to base the evaluation of the quantity of radium in the body on measurements of the radon concentration in the expired breath. Some confusion has resulted from the discrepancies often observed when several breath samples are taken from a given individual by different technics. Studies carried out by the author suggest reasons for these discrepancies and inconsistencies, which have often been mistakenly attributed to faulty analytical technic. It is pointed out that the dilution of exhaled radon depends upon the rate of lung ventilation, which in turn depends upon the rate of metabolic production of CO_2. In consequence of this, it may be possible for two persons, one having a larger surface area than the other, to have different breath radon concentrations, although both have the same amount of radium in the body. This inconsistency may be corrected by calculation of the total twenty-four-hour output of radon from a measurement of the Rn/CO_2 ratio and an estimate of the twenty-four hour production of CO_2 for each individual. The determination of the Rn/CO_2 ratio must be based upon breath samples taken over as long a time as possible and under circumstances which avoid disturbances affecting respiration. Several methods of breath sampling are described, and the results obtained by these methods are discussed. A procedure which avoids effects due to the disturbance of respiration by pressure, resistance, and hyperventilation is suggested.

Significance of Absorbed Radium as Indicated by the Coefficient of Elimination. Frank E. Hoecker. J. Indust. Hyg. & Toxicol. **26:** 289–295, November 1944.

In industry, it has been customary to regard the presence of radium in the body in excess of 0.1 microgram as dangerous and to recommend termination of exposure of the worker until the quantity has been reduced to some very small value. While there is no question regarding the ultimate safety of this procedure, it fails to take into consideration the possibility of normal metabolic elimination of radium during continued employment in the presence of more rigid supervision and improved protective measures.

While 0.1 microgram is considered to be the maximum safe tolerance for radium which is *"fixed"* in the body, the latter term is somewhat ambiguous. Data are presented which indicate that radium is never really *fixed* in the sense that it is metabolically static. Even after as long as twenty years in the body, a small portion continues to be mobile and possible of elimination. The longer it has been in the body, the smaller the rate of elimination becomes. This being the case, it would seem that the significance of the term *fixed* could be resolved by defining it in relation to the rate of elimination. Actually, the potential danger of radium in the body appears to depend upon the quantity present and the total period of retention, both of which depend, in turn, upon elimination.

Important individual differences in the rate of elimination exist. If, therefore, the rate of radium elimination is measured in individual cases where excessive amounts are found, it may be possible to make a more intelligent estimate regarding the potential danger to the individual. That is, an individual whose rate of elimination is high could probably handle radium for many years under proper supervision without accumulating dangerous quantities. This person might be permitted a higher breath radon concentration for a short period of time without its being considered dangerous. On the other hand, it might be necessary to advise another person, showing a low rate of elimination, to change occupation. The individual rate of elimination might also indicate the frequency with which breath samples should be taken.

The following factors may determine the rate of elimination: (*a*) period of absorption, (*b*) rate of absorption, (*c*) mode of entry, (*d*) period of retention, (*e*) metabolism and diet. With respect to past exposure, factors (*a*), (*b*), (*c*), and (*e*) are beyond control. Only (*d*) may be affected by future protective measures. If the past exposure has been excessive, it may be necessary to terminate the absorption period in order to take full advantage of the greatest possible rate of elimination. If the period of absorption has not been too great, it may be possible to alter (*b*) and (*c*), and perhaps even (*e*), sufficiently so that the normal rate of elimination will overbalance the rate of absorption. It may thus be possible to reduce (*d*) for a large portion of the internal radium without terminating exposure.

Supervoltage Roentgen Installation in the Antoni van Leeuwenhoekhuis. Action of Ultra-Hard Roentgen Rays on the Human Skin and on Mice. B. G. van Griethuysen and D. den Hoed. Acta radiol. **23:** 565–567, Dec. 15, 1942. (In English.)

The possible difference between roentgen rays from a 200-kv. apparatus and from a 850-kv. apparatus was investigated by the authors by measuring the lethal dose in mice. A 40 per cent higher dose had to be given with the roentgen rays from the 850-kv. apparatus to kill the mice. It is uncertain whether this difference is due to a somewhat different biological action or to dosimetric factors. MAX SGALITZER, M.D.